A STUDY ON PULMONARY FUNCTION TESTS IN TYPE 2 DIABETES MELLITUS

Dissertation submitted in partial fulfillment of
the requirements for the degree of

M.D. (GENERAL MEDICINE)

BRANCH – I

DEPARTMENT OF GENERAL MEDICINE

KILPAUK MEDICAL COLLEGE,

CHENNAI – 600 010.

THE TAMIL NADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL - 2013

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY ON PULMONARY FUNCTION TESTS IN TYPE 2 DIABETES MELLITUS"** is a bonafide work performed by **Dr.MANOJ KUMAR., P.,** post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2010 to April 2013.

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DECLARATION

I solemnly declare that this dissertation **"A STUDY ON PULMONARY FUNCTION TESTS IN TYPE 2 DIABETES MELLITUS"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision **of Dr.D. Surendran M.D.**, **D.Ch.,** Professor of internal medicine and Unit chief, Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai

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ACKNOWLEDGEMENT

At the outset, I would like to thank my bellowed Dean, Kilpauk Medical College **Prof. Dr. P. Ramakrishnan, M.D., D.L.O.,** for his kind permission to conduct the study in Kilpauk Medical College. I would like to express my special thanks to former medical superintendent **Prof. Dr. Jayaraman, M.S., Mch.,** for permitting to conduct this study in Kilpauk Medical College Hospital.

It gives me immense pleasure to express my sincere and deep gratitude to **Prof. Dr. Gunasekaran M.D.,DTCD.,** Professor and Head of the Department of Medicine, Kilpauk Medical College and Hospital for rendering permission to do this dissertation.

I would like to thank wholeheartedly**, Prof. Dr. Surendran M.D., D.Ch.,** my unit chief and Professor of Medicine for his encouragement and guidance during the study.

With extreme gratitude, I express my indebtedness to **Prof. Dr. Madhavan M.D.,Dip.Diabetology,** Professor and Head, Department of Diabetology, Kilpauk Medical College Hospital for his continuous motivation, timely advice and valuable criticism which enabled to complete the dissertation.

I also express my special thanks to **Prof. Dr. G.Balan., Prof. Dr. S. Usha Lakshmi M.D., Prof. Dr. Ravindran M.D., DNB., Dip Diabetology.**

I am extremely thankful to Assistant Professor of Medicine **Dr. Venkateswaralu M.D., D.Ch** for their assistance and guidance.

I would always remember with extreme sense of thankfulness for the co-operation and criticism shown by my fellow post graduate colleague and friends.

I would like to take this opportunity to show gratitude to my mentor and my dear Father and Mother, my bellowed brother P.Raj mohan, my friends Dr. Ganesh Aravind, Dr. M.Dhanasekar & Dr.Deepa avadhani for their never ending support in finishing this thesis and Almighty God for all his blessings.

Finally, I wholeheartedly thank all my patients for their active co-operation in this study, without which this would not have become a reality.

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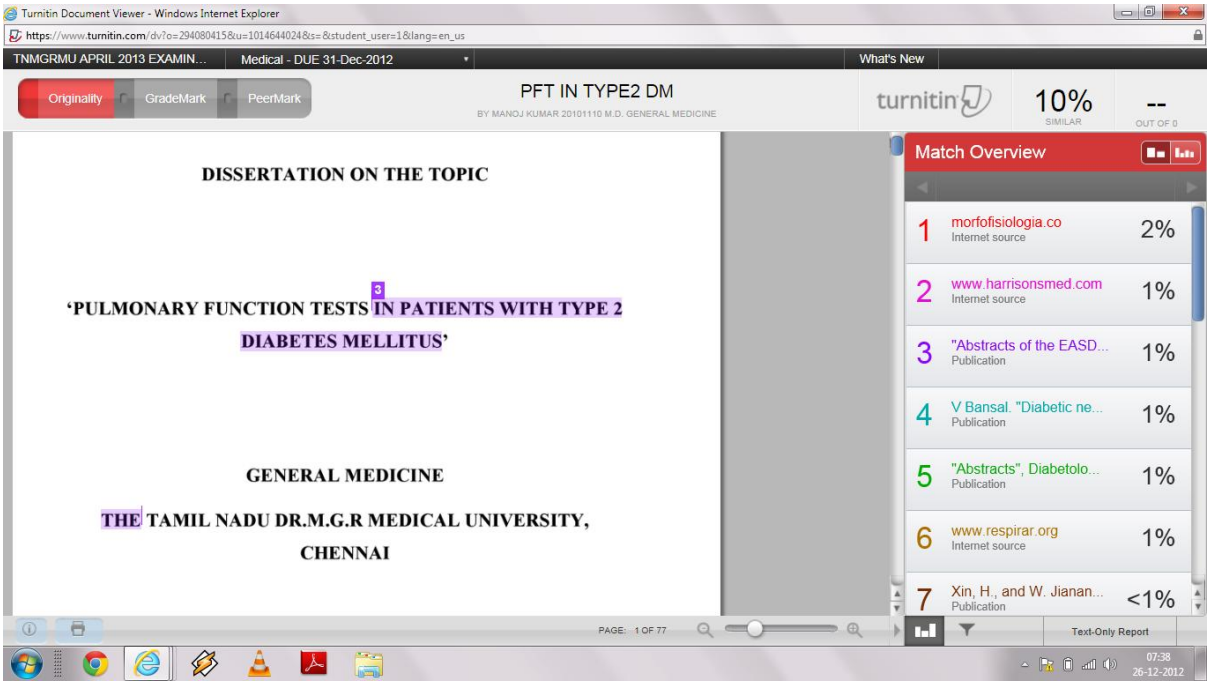
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DISSERTATION ON THE TOPIC
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INTRODUCTION

Diabetes mellitus as we all know is a systemic disorder which affects many organs by causing pathological changes in them. It is considered as a leading cause of increasing morbidity and deaths in todays world.

Many theories have been suggested to explain the end organ damage induced by hyperglycaemia. These are : [1]

1.Formation of advanced glycosylation end products

2.Glucose metabolism via sorbitol pathway

3.Activation of protein kinase C and

4.Increase in flux via hexosamine pathway.

These processes plays the main role in causing impairement of collagen & elastin cross linkage, which therby causes reduced elasticity of connective tissue [2,3] . As per the epidemiological survey, the prevelance of diabetes mellitus is projected to be highest by 2025 in asian Indians [4].

The vascular complications of DM remains the main cause of mortality & morbidity. It includes macrovascular and microvascular ie.., retinopathy, neuropathy and nephropathy. Generally the microvascular complications occur early in the course of diabetes ie.., 5 to 10 years and it takes 10 to 15 years for macrovascular complications to develop[5]. Eventhough respiratory tract infections like tuberculosis is increased in incidence in patients with diabetes mellitus, attention is only minimally given to respiratory system.

This study is aimed to detect the abnormalities in pulmonary functions in Type 2 DM patients. The presence of abundant connective tissue and microvascular circulation in lung raises the possibility that in diabetic patients, lung could also be a "target organ".Chronic hyperglycaemia causes many histopathological changes in the lungs of diabetics. These are thickening of alveolar epithelium and the basal lamina of pulmonary capillaries. These changes ultimately results in reduction in elastic recoiling capacity and the lung volume. The reason would probably be the non-enzymatic glcosylation induced connective tissue alteration in lung parenchyma. [6]

The literature tells us that only very few studies have been carried out to explain the pathological effects of diabetes mellitus on pulmonary functions. So this study would add more to the growing literature on pulmonary functions in patients with diabetes mellitus.

AIM OF THE STUDY

1. To record the Pulmonary function tests in Type 2 DM group as well as in the control group.

2. To compare the pulmonary functions between the 2 groups.

3. To analyse the pulmonary functions in Type 2 DM patients with respect to the duration of diabetes and their recorded blood sugar values.

REVIEW OF LITERATURE

INTRODUCTION :

The group of metabolic disorders which share the phenotype of hyperglycaemia is referred as DIABETES MELLITUS. Complex interaction of various factors including environmental & genetic is the cause for several distinct types diabetes mellitus.

The factors contributing to hyperglycaemia are decrease in insulin secretion, decrease in utilization of glucose, and increase in glucose production. This dysregulation causes secondary changes in various organ systems and imposing burden on the person with diabetes and also on the health care system.[7]

EPIDEMIOLOGY:

The international diabetes federation according to current trends projects that 438 people will have diabetes by the end of year 2030. The rising prevelance of Type 2 DM is more rapid because of sedentary life style, increasing obesity and the aging of population. Geographic variation has a considerable role in the incidence of Type 1 & 2 DM.[7]

The prevelance of Type 2 DM in asia is increasing rapidly and more importantly, the diabetes phenotype appears to be different from the united states – onset at a lower BMI, greater visceral adiposity, younger age and decreased capacity of insulin secretion[7]. People of south asia have a very high rate of diabetes and they too have highest rates of premature CVD (cardiovascular disease). in the world[8].

India, china along with middle east countries are now the 'hot spots' according to diabetes data, since the incidence of disease in next 20 years is likely to double[9]. A rough estimation of prevelance of diabetes is about 12% in india and it is expected to increase in next coming years[10].

According to data from International diabetes federation, 4.6 million people of the age group 20-79 years died in 2011 due to diabetes. This number equals the magnitude of combined deaths from various major infectious diseases. Highest numbers being from india, china & united states of america[11].

There is 13.3% increase in number of deaths related to diabetes in 2011 compared to the estimated deaths of year 2010[12,13].

DIAGNOSIS[7] :

TABLE 1 : DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS
➢ Symptoms of diabetes + RBS ≥ 11.1 mmol/dl (200 mg/dl)[a] OR
➢ FBS ≥ 7.0 mmol/dl (126 mg/dl)[b] OR
➢ HbA1c > 6.5 % OR
➢ Two- hour plasma glucose ≥ 11.1 mmol/dl (200 mg/dl)[c] during OGTT.
[a]RBS(Random blood sugar) is without regard to time since last meal. [b]FBS(Fasting Blood Sugar) after no caloric intake for at least 8 hours. [c]Blood sugar after 2 hours of glucose load containing 75gms of anhydrous glucose in water
Source : American Diabetes Association, 2011

Symptoms of diabetes include :

- Fatigue
- polyuria
- polydypsia
- polyphagia

CLASSIFICATION [7] :

TABLE 2: ETIOLOGICAL CLASSIFICATION OF DIABETES MELLITUS

1. Type 1 Diabetes mellitus (absolute insulin deficiency due to destruction of beta cells)
   - A. Immune mediated
   - B. Idiopathic
2. Type 2 Diabetes mellitus (insulin resistance + insulin deficiency)
3. Other types of Diabetes
   - A. Genetic mutation induced beta cell dysfunction
     - a. Hepatocyte nuclear transcription factor(HNF) 4 alpha (MODY 1)
     - b. Glucokinase---- (MODY 2)
     - c. HNF 1 alpha---- (MODY 3)
     - d. Insulin promoter factor 1---- (MODY 4)
     - e. HNF 1beta---- (MODY 5)
     - f. Neuron D1---- (MODY 6)
     - g. Sub units of ATP-sensitive potassium channel
     - h. Mitochondrial DNA
   - B.Genetic defects in insulin action
     - a. Type A insulin resistance
     - b. Leprachaunism
     - c. Rabson-mendenhall syndrome
     - d. Lipodystrophy syndromes
   - C. Diseases of exocrine pancreas
     - Eg. Pancreatitis, fibrocalculous pancreatopathy, cystic fibrosis, Malignancy
   - D. Endocrinopathies
     - Eg.acromegaly, glucagonoma, pheochromocytoma
   - E.Drug induced
     - Eg.glucocorticoids, thiazides,antipsychotics,hydontoins
   - F.Infections
     - Eg.congenital rubella, coxsackievirus, cytomegalo virus
4. Gestational diabetes mellitus (GDM)

Abbreviation : MODY – Maturity onset ‘diabetes of the young’

INSULIN : BIOSYNTHESIS, SECRETION AND ACTION

BIOSYNTHESIS:

The beta cells of the islets of pancreas produces insulin. The preproinsulin is the initial component which is synthesized as a precursor polypeptide having single chain 86- amino acid. Thereafter undergoes proteolysis to give rise to proinsulin, which is related to IGF 1 & 2 structurally. Further cleavage from proinsulin gives rise to A & B chains of insulin and the C peptide. The beta cells store them together & in secretory granules which are then co secreted[7].

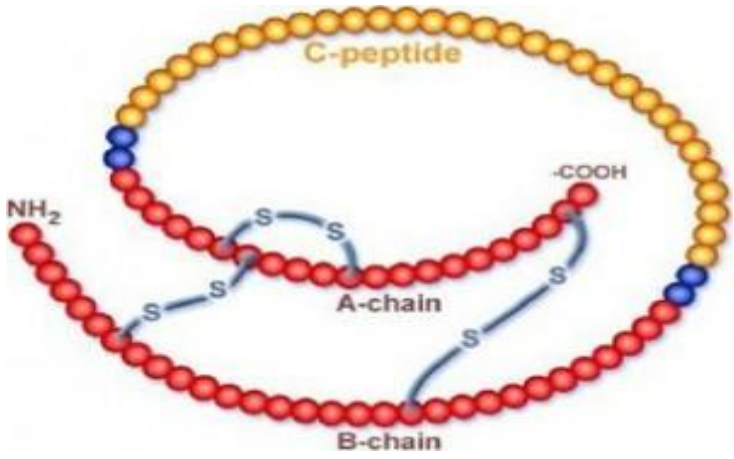


Figure 1 . showing PROINSULIN (A & B chain of mature insulin along with C peptide)

SECRETION:

Insulin secretion by beta cells of pancreas is influenced by many factors like, amino acids, nutrients, ketones, and gastrointestinal peptides. The most important key regulator being glucose. Insulin synthesis is stimulated when glucose levels are above 70mg/dl and primarily due to the enhancement of protein translation and its processing. The rate limiting step which controls the insulin secretion related to glucose regulation is glucokinase mediated glucose phosphorylation. Insulin secretion is stimulated when voltage gated calcium channels open due to depolarization of beta cell membrane induced by potassium channel inhibition[7].

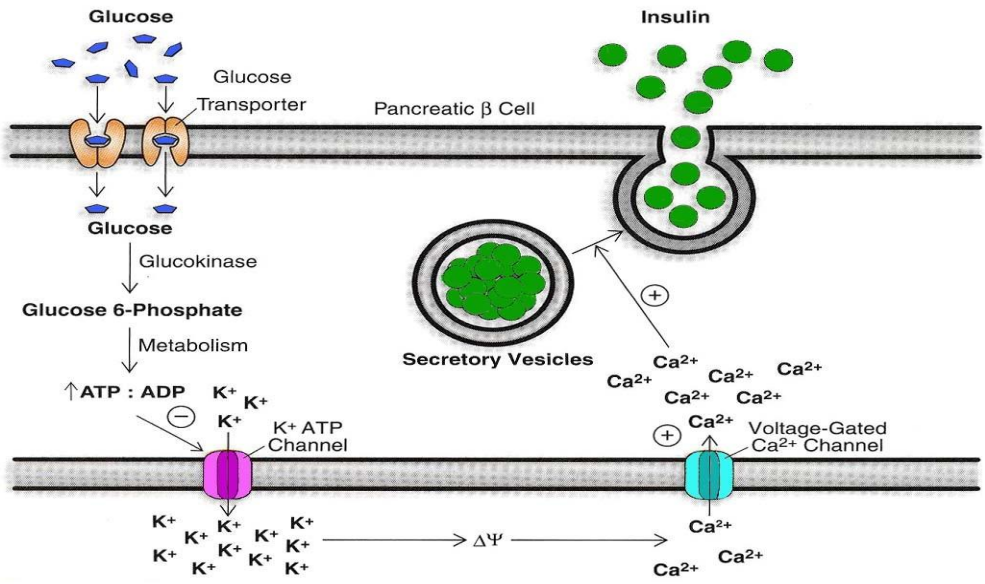


Figure 2. showing mechanism of secretion of insulin.

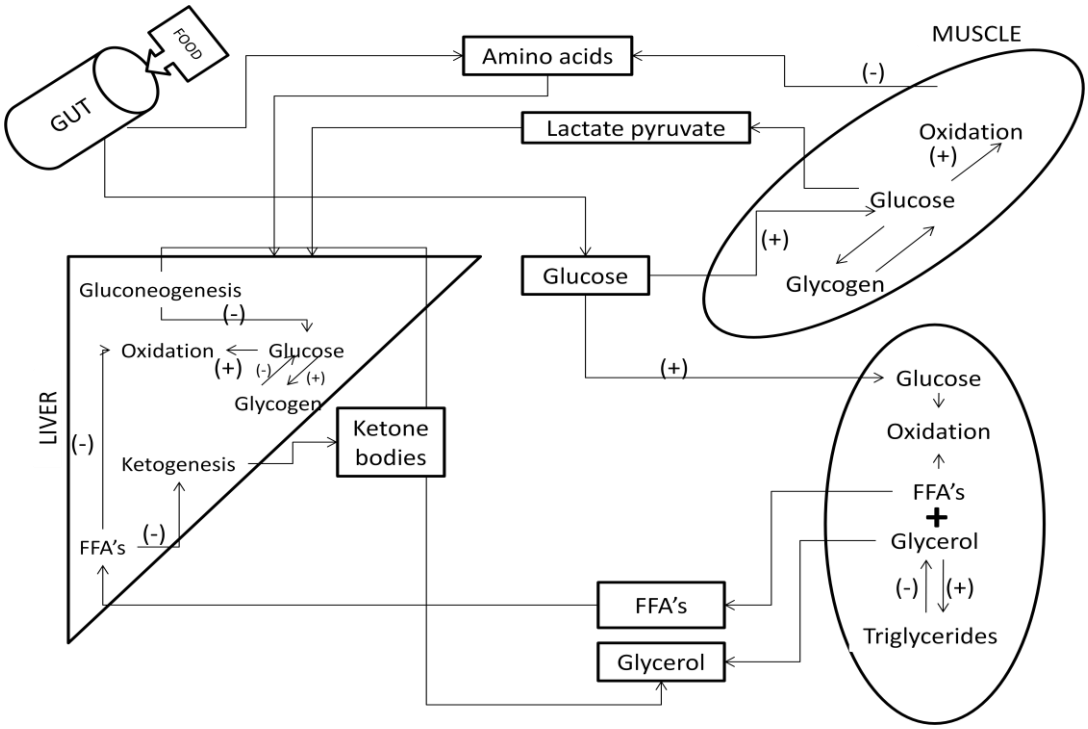
GLUT-2 mediates intracellular transport of glucose. glucose undergoes oxidation to yield ATP which inhibits potassium channel receptor on B cell. thereby causing membrane depolarisation, Ca^2 ion influx followed by release of stored insulin.

Gastrointestinal tract also plays a role by releasing incretins from the neuroendocrine cells when food is ingested and thereby amplifies insulin secretion and suppresses secretion of glucagon. The L- cells of the small intestine release GLP-1 which stimulates secretion usually when blood glucose is more than the fasting level.

ACTION:

Once insulin secretion occurs into the portal venous system, about 50% is immediately removed and undergoes degradation in liver. The remaining enters systemic circulation to bind to target receptors. The receptors gets autophosphorylated along with recruitment of signalling molecules such as IRS i.e., insulin receptor substrates. IRS along with some adaptor proteins initiates a cascade resulting in reaction of phosphorylation & dephosphorylation causing metabolic effects of insulin. Other pathways related to insulin receptor signalling also gets activated to induce glycogen synthesis, lipogenesis and protein synthesis[7].

Figure 3. Metabolic pathway showing fuel metabolism & insulin actions [14]:



(+) ------> stimulation by insulin

(-) -------> inhibition by insulin

FFA ------> Free fatty acids

BLOOD GLUCOSE REGULATION :

Blood glucose regulation is very important for the survival of human beings. Glucose level has to be finely regulated so as to supply glucose to brain which does not have the capacity to store glucose. Glucose also acts as fuel for kidney and RBCs.

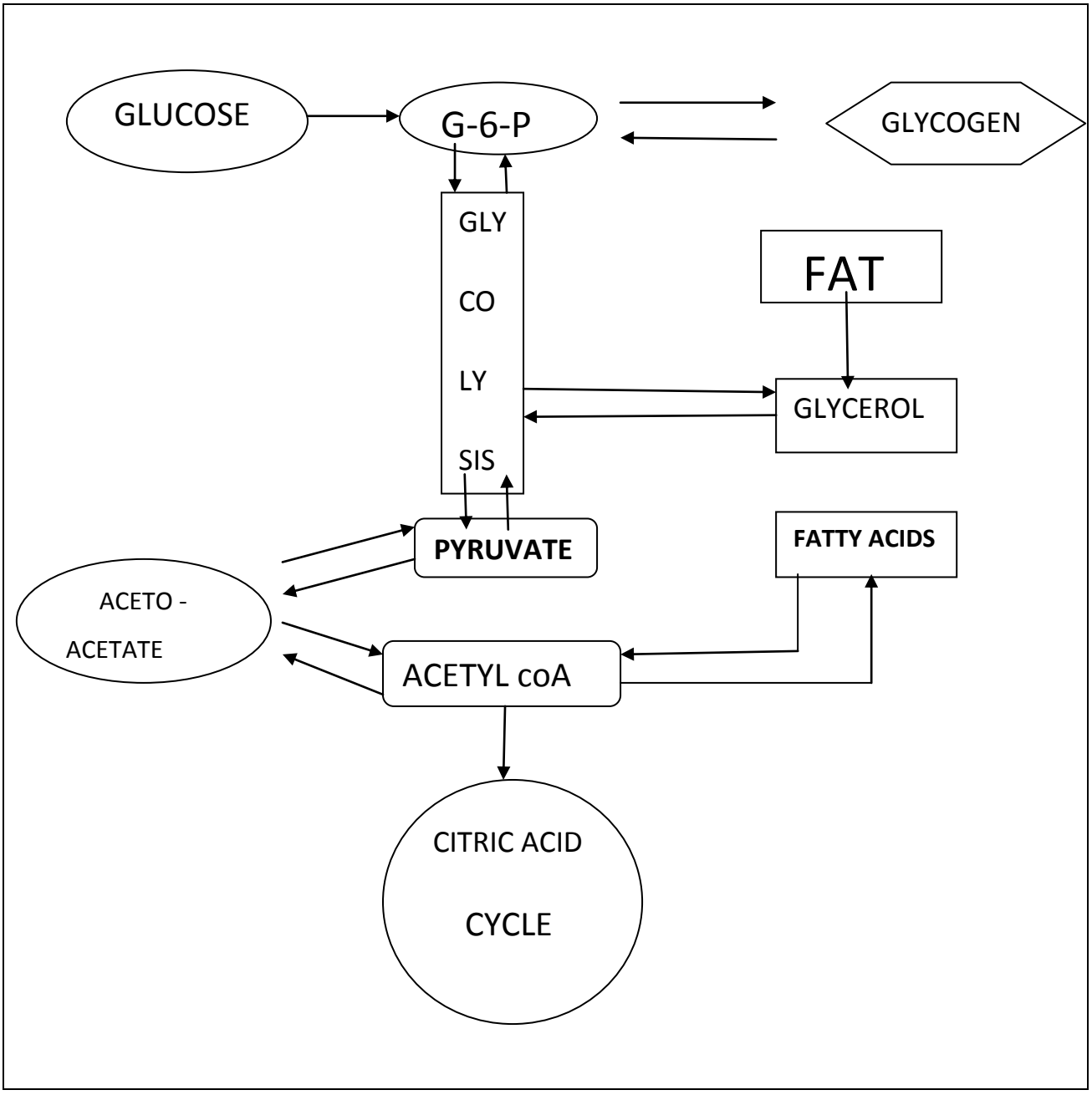


Figure 4. showing glucose metabolism(Glycogenolysis & Glycolysis)

Dietary carbohydrates are finally converted to glucose.[15] Dietary or cellulose protein catabolism results in carbon atoms which are used in glucose synthesis, a process called gluconeogenesis. Tissues other than liver which oxidise glucose incompletely provide lactate which can be converted to glucose through gluconeogenesis[15].

Liver is the predominant organ which responds to fluctuation in blood sugar levels. Synthesis of glucose is on one of the important functions of the liver. Increased or decreased glucose levels trigger several hormonal mechanism that helps to restore normal glucose levels. Whenever there is low blood sugars level, pancreatic a cells are stimulated and glucagon is released[16]. When sugar level is high Beta cells of pancreas are stimulated and insulin is released into the circulation. Pituitary hormones, GH, ACTH inhibit glucose uptake by extrahepatic tissues and thus act to increase blood glucose. Glucocorticoids also inhibit glucose uptake and increased blood glucose levels. Circulating ACTH stimulates adrenal cortex to release cortisol which increases glucose levels in blood. Adrenal medullary hormone, epinephrine which is secreted when there is any stressful event, activates glycogenolysis and increase blood sugar levels[16].

When blood glucose levels are low, there is no uptake of glucose by the liver. Conversely when there is high blood glucose, there will be increased blood glucose uptake by the liver and conversion of glucose to glycogen, stored for future needs.

Whenever there is high blood glucose, there will be high liver glucose. Under such conditions glucokinase activity is increased G6P is rapidly converted to GIP by the enzyme, phosphoglucomutase, which is then incorporated into glycogen[15].

METABOLISM OF GLUCOSE IN DM2:

There will be increased glucose output by liver in DM. First, glycogen is utilized to produce glucose. Next gluconeogenesis takes place. Insulin deficiency causes impairment of glucose utilization by non hepatic tissues. Mainly skeletal muscle and fatty tissue are insulin dependent tissues. Reduced utilization of glucose by peripheral tissue leads to decreased rate of glucose metabolism. As insulin regulates the activity of hepatic glucokinase, there will be decreased rate of phosphorylation of glucose in liver cells leading to increased transport to blood[16]. Combined effect of increased glucose synthesis and reduced utilization leads to elevated plasma sugar values. When renal capacity of sugar handling is overcome by hyperglycemia there will be glucosuria.

Glucose acts as an osmotic diuretic and its loss is accompanied by water and electrolyte loss causing polyuria. Polyuria stimulates thirst mechanism causing polydipsia[16]. Glycosuria, tissue catabolism and impairment of hypopthalamic regulation cause increased appetite and intake of food (Polyphagia)[16].

TYPE 2 DIABETES MELLITUS:

Type 2 DM is characterised by insulin resistance and deficient insulin secretion. The primary defect is still controversial. This systemic disorder has a very strong genetic component[7].

TABLE 3 : GLUCOSE HOMEOSTASIS[7]

VALUE	NORMAL	HYPERGLYCEMIA	
		PRE-DIABETES	DIABETES MELLITUS
		Impaired fasting glucose/ impaired glucose tolerance	
FPG	<100 mg/dl	100 – 125 mg/dl	≥126 mg/dl
2h PG	<140 mg/dl	140 – 199 mg/dl	≥200 mg/dl
HbA1c	<5.6 %	5.7 – 6.4 %	> 6.5 %

Abbreviations: FPG - Fasting Plasma Glucose

2h PG - 2 hrs Post prandial Glucose

HBA1c- Glycosylated hemoglobin

RISK FACTORS FOR TYPE 2 DM [7]:

1. Family h/o DM
2. Obesity (body mass index > 25 Kg/m^2)
3. Race or ethnicity
    a. African American
    b. Native American
    c. Asian American
    d. Latin
4. Presence of impaired fasting glucose, impaired glucose tolerance or HbA1c of 5.7 – 6.4%
5. Systemic hypertension (BP > 140 mm of Hg)
6. HDL cholesterol < 35 mg/dl
7. Triglycerides > 250 mg/dl
8. Polycystic ovarian syndrome or acanthosis nigricans
9. History of cardiovascular disease.

PATHOPHYSIOLOGY:

Impaired insulin secretion, insulin resistance, increased glucose production from liver, and abnormal fat metabolism are the main characteristics of Type 2 DM. Obesity (central or visceral) is strongly associated with Type 2 DM[7].

Initially the beta cells compensate for the insulin resistance in the early stages by increasing insulin output. As this progresses, the beta cells fail to sustain the hyperinsulinemic state. Impaired glucose tolerance develops i.e., elevation in post prandial glucose. Later occurs fasting hyperglycemia due to decline in secretion of insulin and increase in glucose production by liver. Finally there ensues beta cell failure[7].

COMPLICATIONS OF TYPE 2 DIABETES MELLITUS :

ACUTE COMPLICATIONS:

- Diabetic keto acidosis : it results from insulin deficiency which may be relative or absuolute, in combination with excess amount of counter regulatory hormones like cortisol, glucagon, catecholamines, and growth hormone. There occurs promotion of glycogenolysis, gluconeogenesis along with ketone body formation[7].

Hyperglycemia, ketosis along with metabolic acidosis and secondary metabolic derangements in the form of reduction of body stores of sodium, chloride, phosphorous and magnesium. Blood urea and serum creatinine also gets elevated.

- Hyperglycaemic hyperosmolar state : this usually occurs in an elderly patient with Type 2 DM with history of polyuria and weight loss for several weeks. The patient finally ends up in mental confusion and lethargy, or coma.[7] Acute myocardial infarction and Acute cerebrovascular accident being the common cause which precipitates it.

    - Marked hyperglycemia (>1000 mg/dl)
    - Hyper osmolality (>350 mosmol/L)
    - Pre renal azotemia,
    - Ketonemia

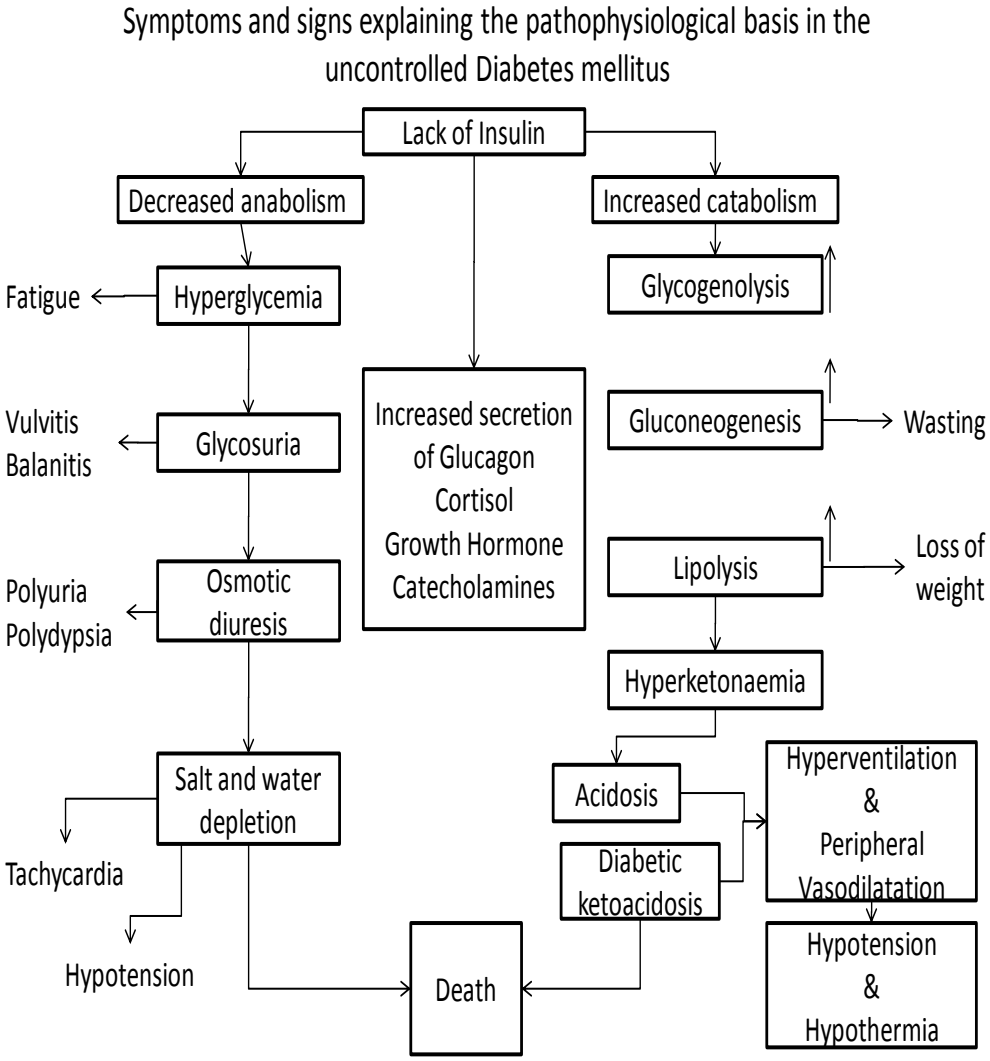


Figure 5. pathophysiological basis for uncontrolled diabetes mellitus.

CHRONIC COMPLICATIONS[7] :

MICROVASCULAR :

1. Eyes – retinopathy (non proliferative/proliferative), macular edema
2. Nerves – neuropathy (sensory/motor, autonomic)
3. Kidneys – nephropathy

MACROVASCULAR :

1. Coronary heart disease
2. Cerebrovascular disease
3. Peripheral arterial disease

OTHERS :

1. Gastroparesis, diarrhea
2. Sexual dysfunction/ uropathy
3. Dermatological
4. Infections
5. Cataract
6. Glaucoma
7. Periodontal disease
8. Hearing loss

MECHANISM OF COMPLICATIONS :

There are atleast four theories, which can explain the chronic complications induced by hyperglycemia.

One theory states that increase in intracellular glucose will lead to AGEs formation, which binds to cell surface receptor. The interaction between glucose and aminogroups on proteins results in non-enzymatic glycosylations. These AGEs can cross link proteins, induce accelerated

atherosclerosis, decrease synthesis of nitric oxide. It also causes endothelial & glomerular dysfunction and alters the extracellular matrix structure & its composition[7].

The second theory states that hyperglycemia in increase glucose metabolism via a special pathway called sorbitol pathway. Metabolism of intracellular glucose predominantly takes place by phosphorylation and glycolysis subsequently. When glucose level increases, some is converted to sorbitol by the action of enzyme aldose reductase. Increase in sorbitol concentration can alter the redox potential therby increasing cellular osmolality, generating reactive oxygen species[7].

The third one states that hyperglycemia activates protein kinase C. This can alter the gene transcription for fibronectin, contractile proteins, type 4 collagen and matrix protein in endothelial cells and neurons[7].

The fourth theory states that there occurs an increase in flux via hexosamine pathway induced by hyperglycemia. It causes function alteration by protein glycosylation or by changing gene expression of TGF beta ie.., transforming growth factor- B or PAI-1 ie.., plasminogen activator inhibitor-1.(7)

GLYCEMIC CONTROL :

The famous DCCT ie.., diabetes control and complications trial was a land mark study which emphasised on the concept that prompt control of chronic hyperglycemia would prevent many complications in case of type1 DM. More than 1400 members were randomized to either conventional diabetes management or intensive management. They were evaluated prospectively for the development of retinopathy, neuropathy or nephropathy.[17,18,19,20]

This study demonstrated that intensive glycemic control can slow down the progression of early complications in diabetes mellitus. Intensive treatment was in the form of multiple administrations of insulin each day along with educational & medical support. Conventional being twice daily insulin injections and quartenary clinical evaluation. [19,20]

Results were:

1. Reduction in NPDR & PDR by 47%
2. Reduction in microalbuminuria by 39%
3. Reduction in clinical nephropathy by 54% & neuropathy by 60%.

The UKPDS ie.., United Kingdom Prospective Diabetes Study covered > 5000 individuals with history of Type 2 DM for > 10 years.

Multiple treatment regimens were utilized for this randomized study ie.., one group with insulin & oral hypoglycaemic agents(sulfonylurea or metformin) and the other group were given conventional pharmacotherapy just to prevent symptoms.[21]

HbA1c of 7% was achieved with intensive treatment arm and 7.9% was in the standard treatment arm. Conclusion was for each percentage reduction in HbA1c there was 35% reduction in microvascular complications.[21,22,23]

Kumamoto study was a small trial study on japanesee with Type2 DM who were lean individuals. Results were reduction in risks of microvascular complications in them. These studies finally gives is an idea about the value of metabolic control in view of reducing the complications[24].

OPHTHALMOLOGIC COMPLICATIONS OF TYPE 2DM:

There are 2 stages of diabetic retinopathy : Non proliferative & proliferative. NPDR is characterised by vascular micro aneurysms, cotton wool spots and blot haemorrhages. The pathophysiology behind is the loss of retinal pericytes, increase in permeability of the retinal

vasculatures, retinal blood flow alteration. These finally lead to retinal ischaemia[14].

Progressing retinal hypoxemia causes neovascularisation which is the hallmark of PDR. These new vessels appearing very near to optic nerve or/and macula can rupture easily causing vitreous haemorrhage and ultimately lading to retinal detachment. If NPDR is severe, it can evolve into PDR within 5 years. Macula edema is clinically significant oly when NPDR is present. Macula edema can be detected by fluorescien angiography. Thereafter moderate visual loss will ensue in few years[14].

Clinical features [14]:

1. MICROANEURYSMS : this is the earliest detected abnormality in most cases. These are tiny and discrete looking circular red dots near to the vessels in retina**.** These microaneurysms look like haemorrhagic spots and they arise from the venous end of capillaries.

2. HAEMORRHAGES : These are round & regular shaped which occur in deeper layers of retina and are called as blot haemorrhages
3. EXUDATES : These are usually seen in the perimacular area with varying size. They also appear as large confluent patches. These are due to leakage of plasma from retinal capillaries which are abnormal.

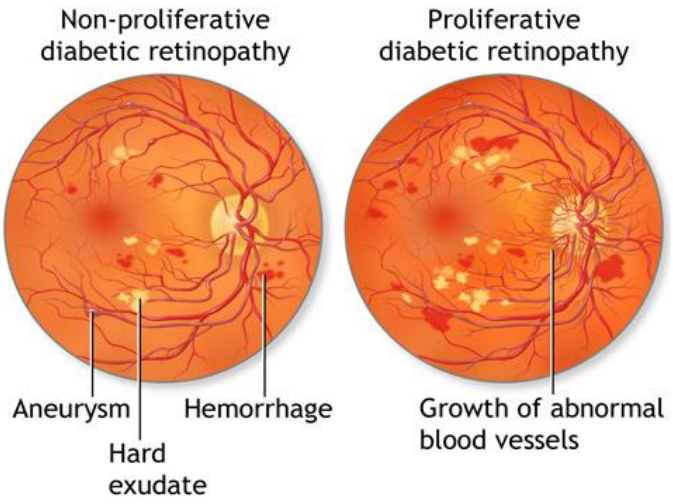
4. COTTON WOOL SPOTS : seen in rapidly progressing retinopathy due to arterial occlusions leading to retinal ischaemia.

5. VENOUS CHANGES : venous dilatation is an early abnormality due to increase in blood flow. Increased tortuosity and beading represents severe form of pre-proliferative retinopathy.

6. NEOVASCULARISATION : New vessels are formed in response to wide spread ischaemia. They arise from the venous circulation forming arcades of tufts of vessels on the retinal surface. They can easily rupture and can cause pre retinal or vitreous haemorrhage. In severe cases causes sudden visual loss.

7. RUBEOSIS IRIDIS : these are new vessels forming on the anterior surface of iris. Its a sign of proliferative retinopathy. Obstruction at the angle of eye can cause secondary glaucoma.

Figure 6 : Fundus picture of diabetic retinopathy



DIABETIC NEPHROPATHY :

Diabetic retinopathy is a leading cause of morbidity and mortality related to diabetes mellitus. The risk of cardiovascular disease also increases in the presence of micro or macroalbuminuria. It is commonly associated with diabetic retinopathy[7,37].

Chronic hyperglycemia leading to ESRD involves[7,38] :

- Effects of soluble factors like AGEs, angiotensin 2, endothelin and growth factors.
- Glomerular hyperfiltration by change in renal microcirculation.
- Change in glomerular structure
- Basement membrane thickening

- Mesangial expansion & fibrosis.

Some of the effects are mediated through angiotensin 2 receptors. The main risk factors is positive family history of diabetes nephropathy. Smoking also causes decline in renal function. The sequential events which are predictable in the natural history of diabetic nephropathy in Type 2 DM appears to be similar to that of Type 1 DM[7].

During the first years after the onset of diabetes , for example in Type 1 DM there occurs an increase in glomerular filtration rate due glomerular hyperperfusion. Over the first 5 years successive changes occur such as glomerular basement membrane thickening along with hypertrophy. Also there occurs expansion of the mesangium. After 5 to 10 years, microalbuminuria sets in and progresses to macroalbuminuria. This proceeds to ESRD in 7 o 10 years [7].

The nephropathy which develops in patients wih Type 2 DM differs from Type 1 in the following ways[7] :

1. Albuminuria (micro or macro) may be present when the diagnosis of Type 2 DM is made.
2. Albuminuria is often accompanied with hypertension.
3. Microalbuminuria cannot always predict nephropathy.

4. Heart failure, hypertension , infections can also cause microalbuminuria which are actually secondary causes.

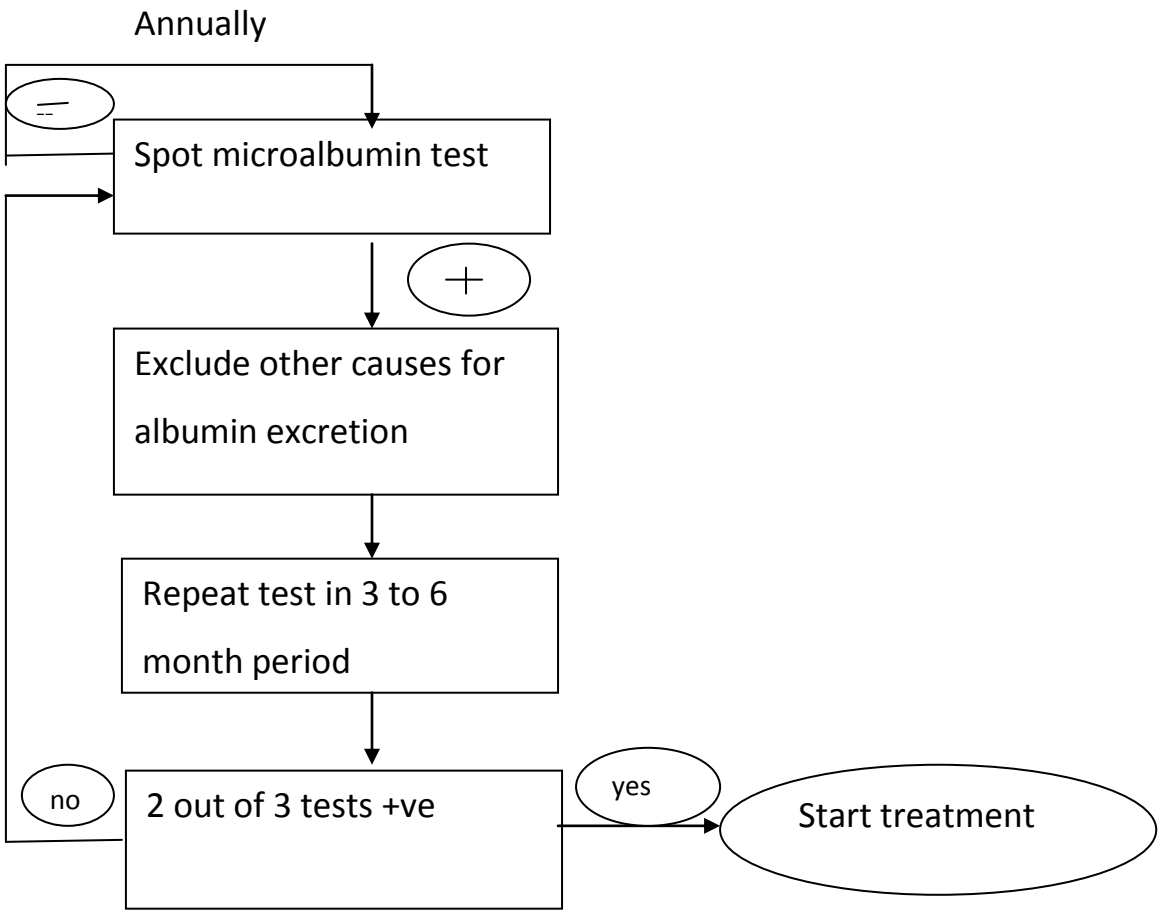
SCREENING FOR MICROALBUMINURIA:

It should be detected at an early stage when therapies can be instituted effectively. Microalbuminuria is an cardiovascular risk factor. Prompt glycemic control can reduce the rate of urine albumin excretion.

The effective interventions for preventing the progression of microalbuminuria to macroalbuminuria are[7]:

1. Glycemic control to normal levels.
2. Very strict control of blood pressure.
3. Starting treatment with ACE inhibitors or ARBs
4. Correction of dyslipidemia.

Figure 7. SCREENING FOR MICROALBUMINURIA



DIABETIC NEUROPATHY

The prevelance of DM in india is 4.3%[25], whereas in west its around 1 – 2%[26]. The reason may be the insulin resistance which is more pronounced in asian Indians[27]. A recent study done in south india revealed 19.1% incidence rate of peripheral neuropathy in Type 2 DM[28]. it is one of the most commonest causes of peripheral neuropathy. Autonomic neuropathy due to diabetes causes death in 25%-50% within a period of 5-10 years[29,30]. In an another study, an increase in incidence of

neuropathy from 7.5% to 50% at 25 years follow up has been documented[31].

CLINICAL CLASSIFICATION OF DIABETIC NEUROPATHY[32] :

SYMMETRIC :

1. Polyneuropathy
2. Painful autonomic neuropathy
3. Painful distal neuropathy with loss of weight known as “diabetic cachexia”
4. Insulin neuritis
5. Polyneuropathy after DKA
6. CIDP with diabetes mellitus

ASYMMETRIC :

1. Radiculoplexoneuropathies (lumbosacral, thoracic, cervical)
2. Mononeuropathies
3. Median neuropthy (wrist)
4. Ulnar neuropathy (elbow)
5. Peroneal neuropathy (fibular head)

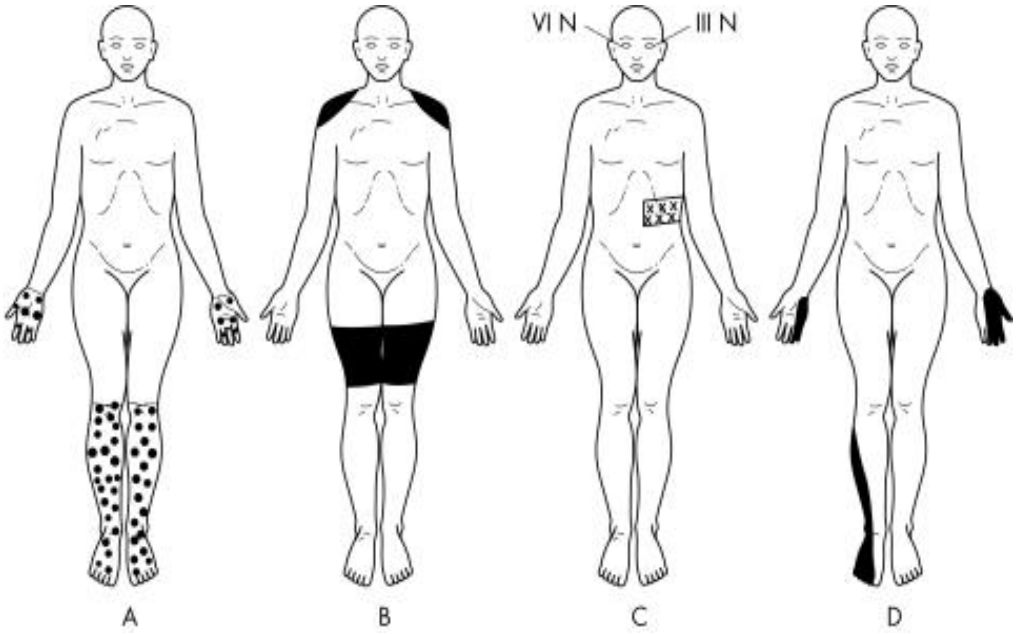


Figure 8. showing various presentations of diabetic neuropathy

A. Distal symmetrical polyneuropathy

B. Proximal neuropathy

C. Cranial neuropathy

D. Mononeuropathy multiplex

Various clinical features of diabetic autonomic neuropathy are :

- resting tachycardia
- orthostatic hypotension
- bladder emptying abnormalities
- sympathetic nervous system dysfunction (hyperhidrosis, anhidrosis)
- hypoglycemic unawareness.

PATHOGENESIS OF DIABETIC NEUROPATHY :

Hyperglycemia causes increased endothelial vascular resistance and reduce the nerve blood flow. It also decreases the level of myoinositol in nerve fibres. Accumulation of fructose and sorbitol in nerve leads to non-enzymatic glycosylation of nerve proteins. Protein kinase C activation also plays a major role in vascular damage to nerve resulting in defective axonal transport[7].

DIAGNOSIS :

It includes assessment of power of muscle, assessment of posterior column and temperature sensations. Tuning fork of 128 Hz is used for testing vibration sense and microfilament of 1 gm for testing touch sensation[33]. The tests based on blood pressure and heart rate response to specific manoeuvres are used to assess autonomic functions. Nerve biopsy is useful just to exclude other causes of neuropathy[34].

The American academy of neurology has recommended that DN should be diagnosed in the presence of sensory or autonomic neuropathy only after excluding other causes[35]. The nerve conduction velocity is diminished gradually in DN, with an estimated loss of about 0.5/sec/year[36].

CARDIOVASCULAR MORBIDITY AND MORTALITY

The very famous FRAMINGHAM HEART STUDY concluded saying increase in the incidence of heart failure, coronary heart disease, peripheral arterial disease and sudden death in diabetes patients. DM has been considered as "CHD risk equivalent" by American heart association. Synergism of increased glucose level with other cardiac risk factor, is the likely the cause of increase in cardiovascular morbidity and mortality rates. The other risk factors include hypertension, obesity, dyslipidemia, physical inactivity, and smoking. Type 2 DM patients have increased levels of PAI-1 (plasminogen activator inhibitor-1) & fibrinogen, which impairs fibrinolysis and enhance the coagulation process[7].

GASTROINTESTINAL AND GENITOURINARY DYSFUNCTION

Gastroparesis and bowel motility alteration are the most common GI symptoms in patients with diabetes mellitus. Symptoms of gastroparesis include nausea, vomiting, abdominal bloating, early satiety. Chronic hyperglycemia causes dysfunction of parasympathetic nervous system and can impair gastric emptying. Normal diarrhea is a feature of GI autonomic neuropathy related to diabetes[7].

The genitourinary abnormalities includes, erectile dysfunction, decreased sexual desire, decreased vaginal lubrication, dyspareunia. Diabetic cystopathy is defined as inability to sense the fullness of bladder & difficulty to void urine comple ely. Later progresses to symptoms like urinary hesitancy and incontinence[7].

INFECTIONS

Diabetic patients have increased frequency and severity of infections. The factors responsible include, defective cell mediated immunity & phagocyte dysfunction due to hyperglycemia, along with decreased vascularization. Some rare infections like rhinocerebral mucormycosis, emphysematous pyelonephritis & gall bladder, malignant otitis externa are excluding seen in Type 2 DM[7].

As far as pneumonia is considered gram negative organism like mycobacterium tuberculosis, staphylococcus aureus are more frequent pathological agents. Escherichia coli is the most common pathogen associated with pyelonephritis in diabetes. DM patients are more to colonization of staph.aureus in the skinfold and nares[7].

DERMATOLOGICAL MANIFESTATIONS

Defective wound healing and ulcerations of skin are common skin manifestations of DM. The pigmented pretibial papules starts with an area of erythema and evolving into circular hyperpigmentation. These results from minor trauma in the pretibial region.[7]

The other specific skin disorders includes[7]

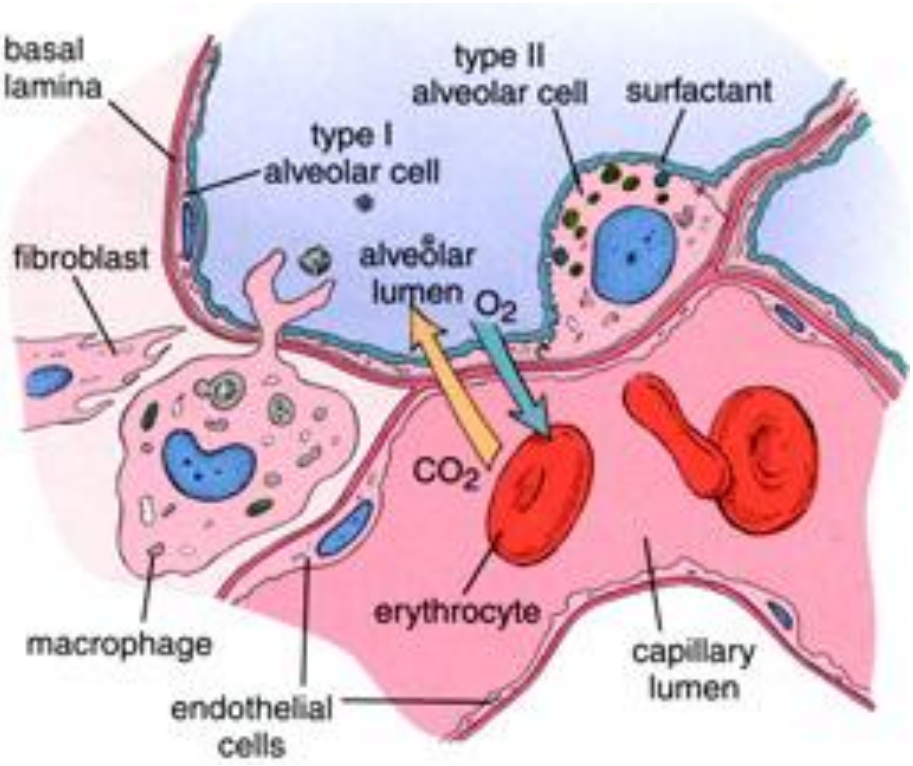
a. Bullosa diabeticorum
b. Necrobiosis lipoidicum diabeticorum
c. Granuloma annularae
d. Sclerederma.

EFFECT OF DIABETES ON RESPIRATORY SYSTEM:

Integrity of connective tissue of the lung and microvasculature of the lung influence the normal mechanics and gas exchange. Diabetes effects either of these two structural components leading to pulmonary function abnormality, like decreased vital capacity, compliance of the lung, TLC, decreased central and peripheral airflows, rapidity of aging process, thickening of capillary endothelial basement membrane and alveoli, modification of surfactant and its function, pulmonary

microangiopathy which leads to decreased diffusing capacity and endurance of respiratory musculature.

Figure 9. showing alveolar structure

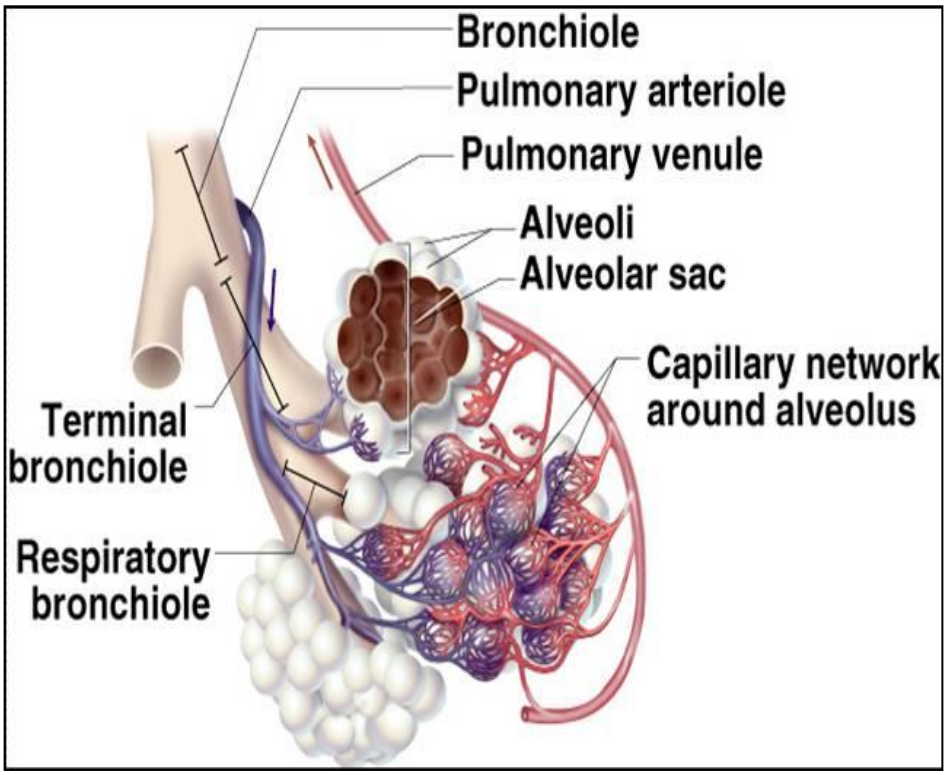


PULMONARY FUNCTION TEST:

Gas exchange remains the most essential fuction of lung, occurring at the alveolar level. ie., between the air in the alveoli and the blood present in the alveolar capillaries. Due to the gas transfer the pressure is maintained for oxygen at 100mm of Hg and for carbondioxide its around 40mm of Hg. This is achieved through various mechanisms like

ventillation, diffusion, oxygen transport and its utilisation. Lungs also play a vital role in maintaining the acid base balance in our body[39].

Figure 10 . showing the bronchopulmonary tree with its vasculature



Assessment of pulmonary functions has a wide range of applications[39] :

1. Characterising the type of dysfunction to aid diagnosis
2. Preoperative evaluation
3. Assessment of disease progression
4. Monitoring response to therapy

Spirometer is a simple instrument which is used to diagnose and manage patients with pulmonary disorders. The interpretation is challenging as it is mainly dependent on patients effort and the interpreter's knowledge about the reference values as per age, sex and weight. A stepwise method is recommended. Determining the validity of the test should be the first step. Next being the determination of ventillatory pattern ie., normal, obstructive or restrictive[39].

TABLE 4. showing spirometric parameters

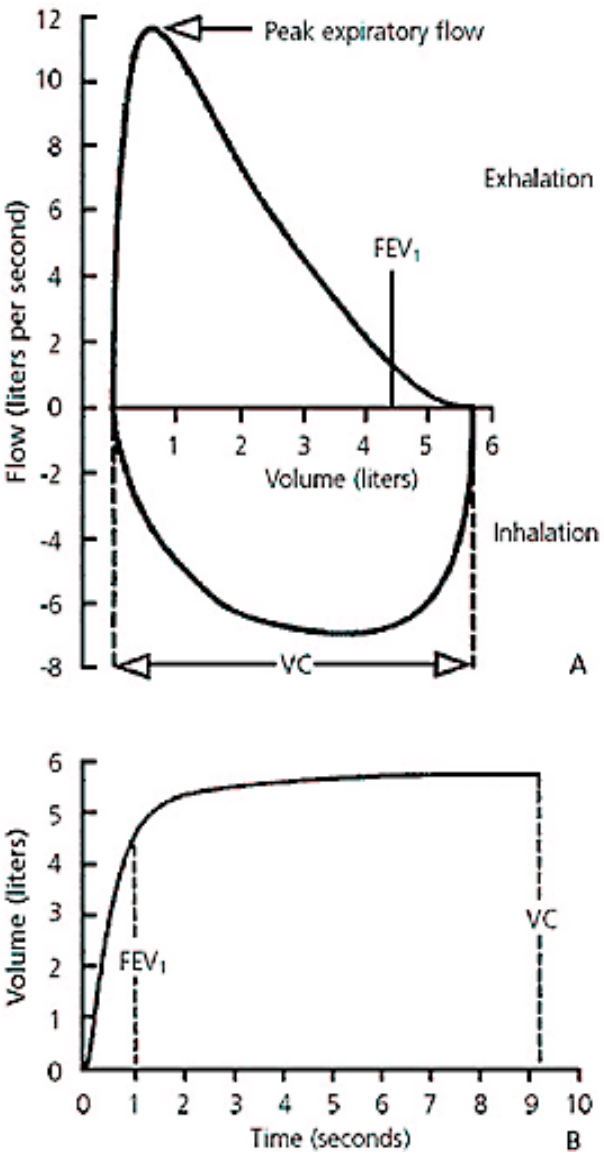
FVC	Forced vital capacity	Litres
FEV1	Forced expiratory volume in 1st second	Litres
FEV1/FVC	The % of FVC expired in 1st second	percentage
$FEF_{25\%-75\%}$	Forced expiratory flow over middle one half of FVC	Litres/min
PEF	Peak expiratory flow rate	Litres/min

Spirometry Measurements

Spirometry records and measures the rate of lung volume changes during forcefull breathing. It begins with a full inhalation, followed by rapid emptying of the lungs by forced exhalation, continuing till a plateau in exhaled volume is attained. These efforts are finally recorded and graphed.

The most important spirometric data is the FVC, the patient inhales maximally, followed by exhaling completely as possible. Normally lungs can empty > 80 percent of their volume in 6 secs or less. The FEV_1 is the volume of air which is exhaled in the 1st second of the FVC maneuver. The FEV_1/FVC ratio is generally expressed as a percentage. For interpretation,the absolute value is used and not the percent predicted[39].

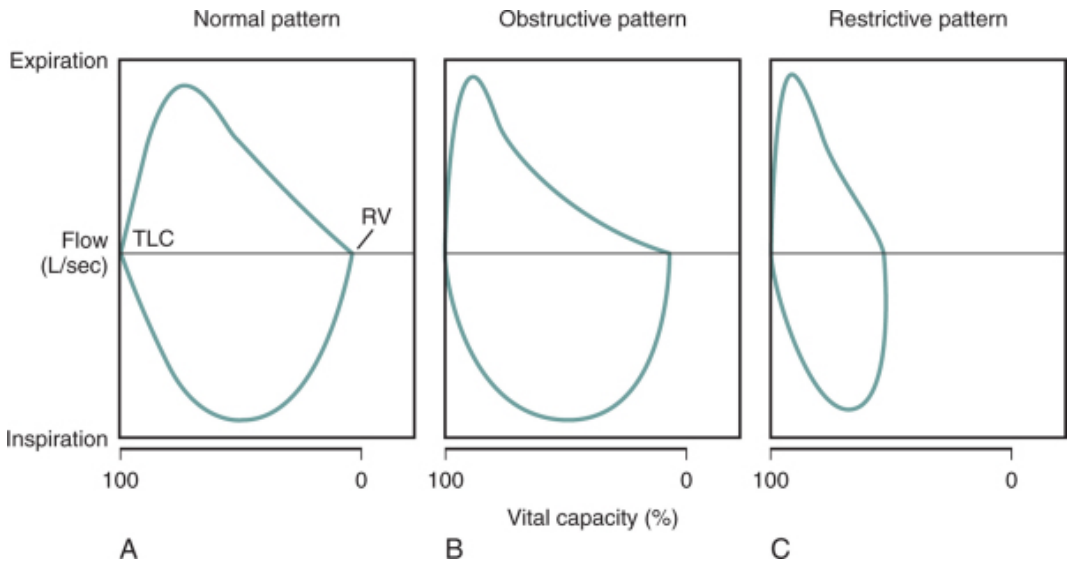
figure 11. normal spirometric flow curve



(A) Flow-volume curve
(B) Volume-time curve extending more than 6 seconds.

Diagram showing normal spirometric flow

Figure 12. patterns of spirometric curves



Interpreting Spirometry Results

Before interpretation , the results must be assessed for validity.[40,41] often there occurs misdiagnosis because of inadequate patients effort. The normal spirometry values vary with respect to patient's age, height, weight, sex, and ethnic background[42,43]. Predicted values may be inaccurate in very tall individuals or persons with absent lower extremities. FEV1 and FVC are lesser in blacks and Asians compared to whites. FVC can vary with the change of position. FVC is about 2 percent greater in standing position compared to supine.

At least 3 acceptable spirograms should be obtained to determine the validity of results. Every time, patient should exhale for at least 6 secs and stop when no volume change occurs for 1 second. The session is over when the difference between 2 largest FVC values and between the 2 largest FEV_1 values is within 0.2 L. If both these are not met even after three times, interpretation is not recommended. Tests should be repeated till the criteria are met[44].

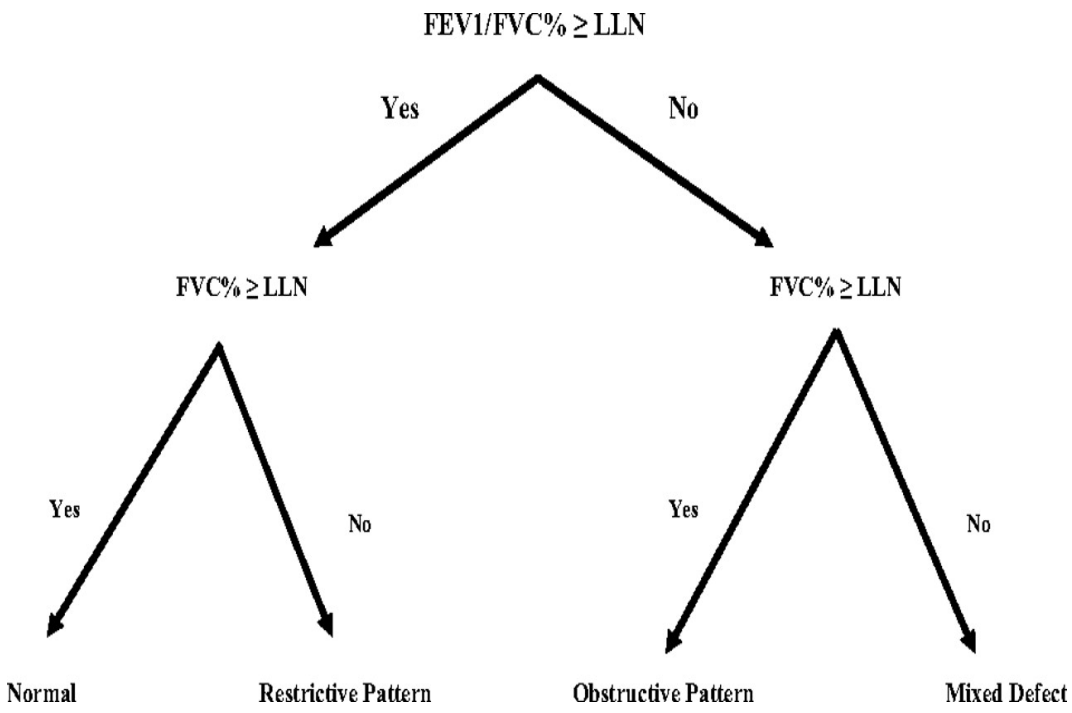
When there is decrease in FVC and FEV1 , it could be obstructive or restrictive but the distinction will depend on the absolute FEV_1/FVC ratio. If ratio is normal or increased, a restrictive defect may be present. However, to confirm restrictive lung disease, the patient should assessed with respect to his lung volumes. If the TLC is<80%, the pattern is restrictive.

A reduced FEV_1 and absolute FEV_1/FVC ratio suggests an obstructive ventilatory pattern. The mid-expiratory flow rate ($FEF_{25–75\%}$) is the average forced expiratory flow rate recorded over the middle 50 % of the FVC. It helps in diagnosing an obstructive ventilatory pattern.usually the reduction in $FEF_{25–75\%}$ of <60 % of the predicted and low to normal FEV_1/FVC ratio may confirm airway obstruction[45].

The final step in interpretation is to determine if any additional

testing is necessary to further define the detected abnormality by spirometry. Static lung volume measurement is required to make a definitive diagnosis of restrictive type of lung disorder.

Figure 13. showing flow chart to interpret spirometric values



HISTORICAL REVIEW ON WORKS RELATED TO PFT IN TYPE 2 DM

P.Lang et al documented the possible associations between Diabetes mellitus, plasma glucose and the spirometric values like FVC & FEV1. They did a cross sectional study covering about 11,763 subjects of Copenhagen city. It showed slight impairement in lung function which was more prominent in diabetic subjects, who were taking insulin than those taking oral hypoglycaemic agents. They finally concluded saying that both

IDDM & NIDDM were having reduced lung functions since they had decreased FVC & FEV1.[46]

There are many studies which have reported decreased pulmonary capacity in DM patients due to changes in elastic properties of lungs.[47,48,49,50]

Walter.E.Robert et al showed the association between state of glycaemia and the pulmonary functions. It included 87 members of Framingham and the result was reduction in FVC, FEV1 & FEV1/FVC ratio.[51]

Davis.A.Wendy et al studied and explained about the concept of reduced pulmonary function test in Type 2 DM in association with their glycemic exposure. This study included 495 patients with Type 2 DM and were studied between 1993 to 1994 by cohort study and 125 were restudied after 7 years. They found a decrease in mean % predicted values and annual decline of FVC, FEV1, PEF and Vital capacity.[52]

Is lung a "target organ" in diabetes mellitus was popular study carried out by Malcolm Sander. There is evidence of lung involvement in diabetic patients which is histopathologically proven ie.., by the process of alveolar epithelial thickening and thickening in the basal lamina of pulmonary capillaries highly suggesting the phenomenon of pulmonary microangiopathy. Probably the alteration in lung tissue is due to the non-enzymatic glycosylation, which thereby causes decreased elastic recoiling of lung tissues. These changes finally leading to reduced lung volumes in diabetes mellitus.[53]

Another study carried out by Davis Timothy M.E et al on pulmonary functions in Type 2 DM covering about 421 patients. It showed reduction in the means of spirometric measurements by 9.5%. They also showed that there was no correlation between HbA1c and

spirometric measures, but there was a correlation with respect to duration of diabetes mellitus.[54]

Sreeja C.K et al studied lung function on 20 subjects with Type 2 DM, 20 with Type 1 DM and 40 control subjects. It showed significant reduction in FEV1/FVC, total lung capacity and lung volumes in both diabetic groups compared to control. They also suggested alteration in collagen tissue of lungs & loss its of recoiling capacity.[55]

Mohan kumar et al studied on pulmonary complications in diabetics of elderly age group. Total lung capacity, lung volume and compliance of lung are decreased. Diffusion capacity of carbon monoxide was also reduced in them. They attributed the presence of increased non-enzymatic glycosylation which interferes with connective tissue cross linkages[56].

Dharwadkar AR et al studied 40 type 2 DM patients with age above 30 years and duration of 1 to 20 years. He showed reduction in lung parameters like FEV1, FEV1% and demonstrated negative correlation with respect to glycemic status[57]

A study by A.S Agarwal et al in 2010 exhibited decrease in diffusion capacity in Type 2 DM patients.[58]

Hsin-chieh et al did a cross sectional study in diabetic patients & prospectively analysed the association with declining lung functions. The diabetic group showed significant reduction in FVC and FEV1.[59]

There are also reports which suggest there is no correlation between lung functions and the presence of microvascular complications or the status of glycaemic control.

MATERIALS AND METHODOLOGY

45 Type 2DM patients were selected for this study of age more than 35 years and with duration of diabetes > 5 years. These patients were selected from Dr.Ambedkar Institute Of Diabetology, KMCH (kilpauk medical college hospital) who were getting treated as out patients. 45 healthy volunteers were assigned as control group, most of them being staffs of kilpauk medical college hospital.

This study was a randomised case control study carried out from January 2011 to December 2011,in collaboration with departments of Diabetology, Respiratory medicine, & Department of ophthalmology. The exclusion criterias were Smokers, history of hypertension, obesity, previous history of lung disease (COPD, PTB, PT SEQULAE), Subjects with signs & symptoms of respiratory infection at the time of test, Subjects having history of admission for respiratory illness during past 6months & Subjects with cardiovascular illness.

The study was carried out after explaining the procedure & taking a written informed consent from the patients and volunteers.

DESCRIPTION OF THE DEVICE :

"SPIROLAB" is a new generation spirometer. It facilitates the total valuation of lung functions. This apparatus can measure FVC(forced vital capacity), VC(slow vital capacity), MVV(maximum voluntary ventilation test) and breathing pattern tests and calculates an index of test acceptability and a measure of reproducibility , and also gives functional interpretation following the latest American thoracic society classification.

Figure 14 showing SPIROMETER:

This apparatus is supplied with an RS-232 opto-isolated serial communication port. The core of the system is a flow meter which is a bi-directional digital turbine flow meter which is connected through the serial port RS-232 to a data elaboration device that use electronic and mechanical procession components.

The spirometric parameters are measured and displayed after each test. The flow/volume curve is shown in real time. Each time can be repeated several times. The best parameters are used for interpretation. After each test session, the results are compared to the relevant predicted values and the percentage ratio between measured and predicted is shown for each parameter. The predicted values are selected as recommended by ERS (European Respiratory Society).

% predicted = measured/predicted × 100

The test can be repeated more than once and the best result is memorized in order to recall it from the spirometer's memory. The best test is determined following the ATS(American Thoracic Society) and ERS(European Respiratory Society) standards.

MEASURING HbA1c :

HbA1c is a measure of the quantity of glucose which is attached to the haemoglobin present in red blood cells. The higher the A1c, the higher was the blood glucose levels over the last two to three months. HbA1c was measured for the study group using "BIO-RAD" method.

MEASURING FOR MICROALBUMINURIA :

Microalbuminuria was measured in a spot urine sample collected from the patient. The interpretation about the presence was made on basis of ADA recommendations. The method used was NEPHALOMETRY.

NORMAL	< 30 mg/g creatinine
MICROALBUMINURIA	30-299 mg/g creatinine
NEPHROPATHY	>300 mg/g creatinine

STATISCAL ANALYSIS: The SPSS software was used to analyse the obtained datas statistically. Various tests and methods were implemented to analyse such as student 't' test , pearson's correlation coefficient test etc. Microsoft excel and word were used to compute the details.

SELECTION OF GROUPS :

The diabetics were selected from diabetology out patient clinic. All were of age more than 35 years and with more than 5 years duration of diabetes. An elaborate medical history was obtained through a questionnaire which covered patients general details, smoking history, family history, treatment history to rule out the exclusion criterias. Height, weight and BMI, waist circumference was recorded. They were screened for hypertension as per JNC 7 blood pressure guidelines[JNC 7]. Blood sample for measuring FBS, ECG to rule out ischaemic heart disease, chest xray to rule out COPD and other respiratory illness like tuberculosis (new or old) were taken. Volunteers for the control group were selected in the same above said way.

BMI:

BMI was calculated as weight(in kgs) / height ^{2} (in meters).

BMI>30 were excluded from the study.

BLOOD PRESSURE :

Blood pressure was measured for the groups using a mercury sphygmomanometer in right upper limb in supine supine position. For the

diabetic group, the measurement in standing position was also made to find out postural hypotension.

WAIST CIRCUMFERENCE :

The waist circumference was measured from the midpoint between the lower ribs measured at the sides and the iliac crest. This was done using a measuring tape.

PROCEDURE OF LUNG FUNCTION TESTING :

The procedure of spirometry test was demonstrated to both the groups and the test was done for them after obtaining written consent from them. The test was done using a RS 232-C spirometer. It was repeated 3 times at an interval of 15 minutes. The following values were recorded:

- FVC
- FEV1
- FEV1/FVC
- PEF
- FEF 25%-75%

Printouts of the recorded values were taken from the spirometer for analysis.

PROCEDURE :

Figure 15. showing how spirometry is done

The best of 3 values were taken and interpretation was made.

After 2 hours, blood sample was taken for measuring PPBS and HbA1c levels. Urine was tested for the diabetic group for the presence of albumin. The diabetic patients were shown to an expert ophthalmologist in KMCH to check for the presence of diabetic retinopathy.

The details of all the 45 diabetics and 45 controls were tabulated as master charts. Statistical treatment was given by the following:

1. Student ‘t’ test which was used to find out the significant difference of pulmonary functions between the study and control group.

2. Pearsons correlation coefficient[60] was made use to find out the correlation of spirometric values with respect to duration of diabetes, FBS, PPBS, HbA1c.

If r = +.70 or higher--- Very strong positive relationship

+.40 to +.69 ----Strong positive relationship

+.30 to +.39 ----Moderate positive relationship

+.20 to +.29 ----weak positive relationship

+.01 to +.19---- No or negligible relationship

-.01 to -.19----No or negligible relationship

-.20 to -.29 ----weak negative relationship

-.30 to -.39---- Moderate negative relationship

-.40 to -.69---- Strong negative relationship

-.70 or higher---- Very strong negative relationship

RESULTS AND ANALYSIS

Spirometry was done in 45 Type 2 DM patients & 45 healthy volunteers. The aim was to find out the effect of diabetes mellitus on lung functions.

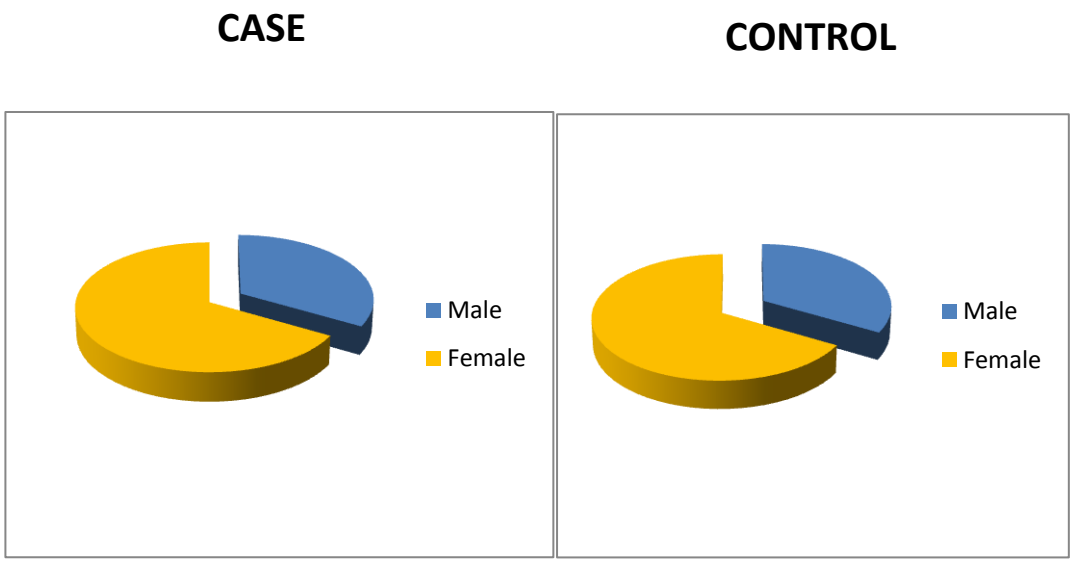
The basis characters of both the groups are shown in Table. 6. The results of the spirometry test for both the groups are shown in Table- & Table-. Rearrangements of the master charts were done according to the requirements to find out the relationship of variables like duration of DM, FBS, PPBS, HbA1c on the pulmonary functions.

Student ‘t’test was used to analyse the statistical difference of the spirometric values between the 2 groups.

Pearson’s correlation coefficient test was used to find out the statistical correlation of spirometric values with duration of DM, FBS, PPBS, HbA1c .

Table 5. showing sex distribution in case & control groups

Group	Sex	Frequency	%
Case	Male	15	33.3
	Female	30	66.7
Control	Male	15	33.3
	Female	30	66.7



Both the groups (CASE & CONTROL) had 15 males and 30 females. They were of age more than 35 years with a BMI of less than 30.

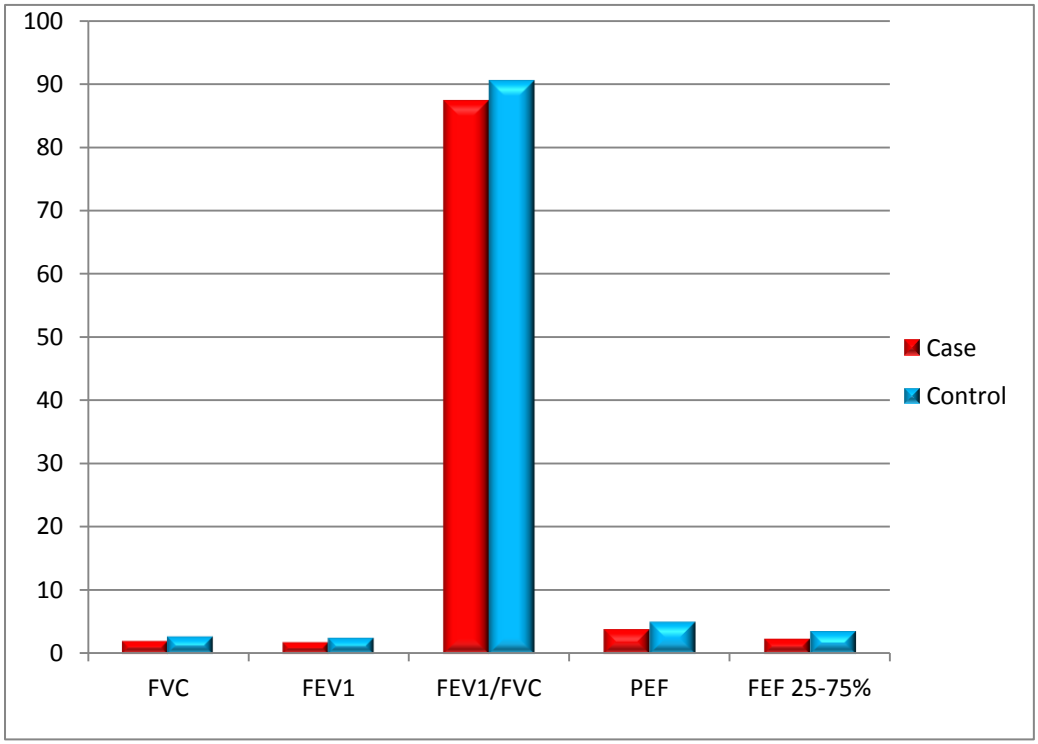
Table 6. showing comparison of spirometric values of the 2 groups

Parameters	Case			Control			'p' value
	Mean	Range	SD	Mean	Range	SD	
FVC	2.09	1.41-3.21	0.55	2.8	1.72-3.93	0.63	0.000
FEV1	1.87	0.95-3.33	0.54	2.6	1.67-5.35	0.7	0.000
FEV1/FVC	87.38	50.3-96.1	7.16	90.58	77.9-94.1	2.77	0.006
PEF	3.96	1.6-7.81	1.6	5.1	2.43-8.34	1.76	0.002
FEF 25-75%	2.41	1.03-5.62	1.04	3.63	1.94-5.36	1.02	0.000

Impact of Type 2DM on Pulmonary functions

The impact of Type 2DM on pulmonary functions is shown in Table. 6. The statistically significant difference was made out between the 2 groups using student ‘t’ test.

Graph 1.showing difference in spirometric values between 2 groups



Type 2DM and FVC :

Table - & graph – reveals that the FVC in Type 2 DM is decreased when compared to the control group. Type 2 DM patients having a mean at 2.09±0.55 with a range of 1.41-3.21 compared to controls having a mean of 2.80±0.63 with a range of 1.72-3.93.

This result favours the concept of Davis M.E Timothy et al's study which showed that FVC in diabetics is decreased by 9.5%.

Type 2DM and FEV1 :

Table - & graph – reveals that the FEV1 in Type 2 DM is decreased when compared to the control group. Type 2 DM patients had a mean at 1.87±0.54 with a range of 0.95-3.33 compared to controls having a mean of 2.60±0.70 with a range of 1.67-5.35.

This result also favours the concept of Timothy's study who showed an average of 9.5% decrement of FEV1 in diabetic patients.

Type 2 DM and FEV1/FVC :

Table - & graph- shows that there is little difference in FEV1/FVC in the form of decrement when compared to the controls, which is of statistical significance.

Type 2 DM patients had a mean at 87.38±7.16 with a range of 50.3-96.1 compared to controls having a mean of 90.58±2.77 with a range of 77.9-94.1.

Type 2 DM and PEF :

Table - & graph – reveals that the PEF in Type 2 DM is decreased when compared to the control group. Type 2 DM patients having a mean

at 3.96±1.60 with a range of 1.6-7.81 compared to controls having a mean of 5.10±1.76 with a range of 2.43-8.34.

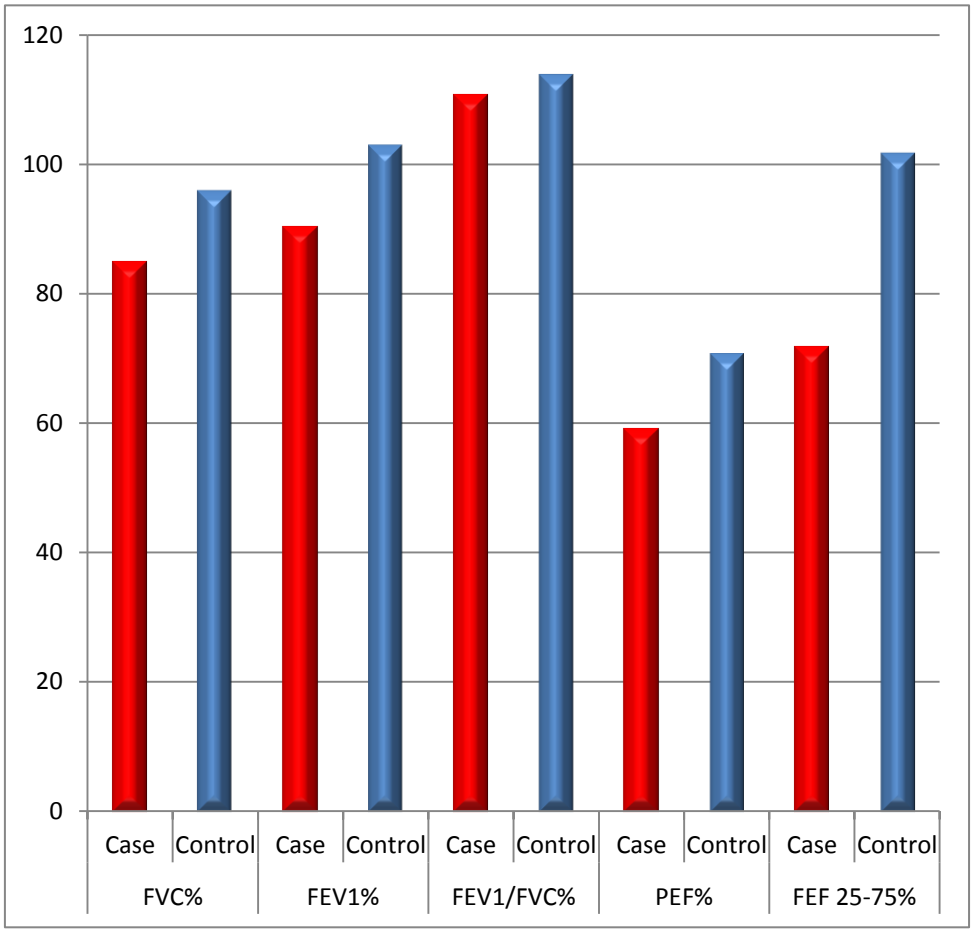
Type 2 DM and FEF 25-75% :

Table - & graph – reveals that the FEF25-75% in Type 2 DM is decreased when compared to the control group. Type 2 DM patients having a mean at 2.41±1.04 with a range of 1.03-5.62 compared to controls having a mean of 3.63±1.02 with a range of 1.94-5.36.

Table 7. showing the difference in % prediction between 2 groups

Parameters	Group	Mean	S.D	'p' value
FVC%	Case	84.84	17.05	0.000
	Control	95.87	9.47	
FEV1%	Case	90.24	16.62	0.000
	Control	102.91	10.63	
FEV1/FVC%	Case	110.69	8.87	0.034
	Control	113.8	3.89	
PEF%	Case	58.96	18.43	0.001
	Control	70.76	13.72	
FEF 25-75%	Case	71.64	23.11	0.000
	Control	101.67	19.87	

Graph 2. showing the difference in % prediction between 2 groups



Type 2 DM and FVC%

FVC% is decreased in diabetic group when compared to the controls. The mean in diabetic group being 84.84±17.05 when compared to 95.87±9.47 in control group.

Type 2 DM and FEV1%

FEV1% is decreased in diabetic group when compared to the controls. The mean in diabetic group being 90.24±16.62 when compared 102.91±10.63 in control group.

Type 2 DM and FEV1/FVC%

FEV1/FVC% is decreased in diabetic group when compared to the controls. The mean in diabetic group being 110.69 ±8.87 when compared to 113.80 ±3.89 in control group.

Type 2 DM and PEF%

PEF% is decreased in diabetic group when compared to the controls. The mean in diabetic group being 58.96 ±18.43 when compared to70.76 ±13.72 in control group.

Type 2 DM and FEF 25-75%

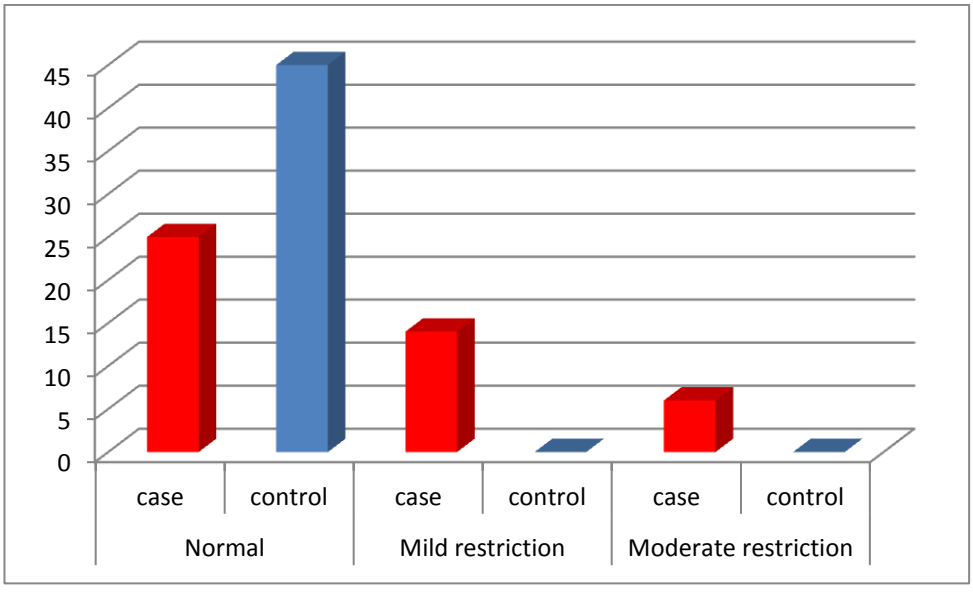
FEF 25-75% is decreased in diabetic group when compared to the controls. The mean in diabetic group being 71.64 ±23.11 when compared to 101.67 ±19.87 in control group.

Table 8. DISTRIBUTION OF SPIROMETRIC

PATTERN IN TYPE 2 DM

Spirometric pattern		Case	Control
Normal	Count	25	45
	% within interpretation	35.70%	64.30%
Mild restriction	Count	14	0
	% within interpretation	100%	0
Moderate restriction	Count	6	0
	% within interpretation	100%	0

Graph 3. showing pattern of lung function in diabetic group



- ❖ 31% of the diabetics had mild restriction of pulmonary functions
- ❖ 13% of the diabetics had moderate restriction of pulmonary functions.
- ❖ These collectively had long duration of DM compared to rest of the diabetics who underwent spirometry test.

TABLE 9. CORRELATION OF PFT VALUES WITH DURATION OF DIABETES

FVC	FVC-R	-0.280
	FVC%	-0.445**
FEV1	FEV1-R	-0.259
	FEV1%	-0.498**
FEV1/FVC	FEV1/FVC-R	-0.253
	FEV1/FVC%	-0.209
PEF	PEF-R	-0.296*
	PEF%	-0.471**
FEF 25-75%	FEF 25-75%-R	-0.297*
	FEF 25-75%(%)	-0.461**

**. Correlation is significant at the 0.01 level (2-tailed).

FVC & Duration of Diabetes

FVC-R has a weak negative correlation with respect to duration of diabetes. FVC% has more strong negative correlation with respect to duration of diabetes, which is more significant, ie.., FVC decreases with increasing duration of diabetes.

FEV1 & Duration of Diabetes

FEV1-R has a weak negative correlation with respect to duration of diabetes. FEV1 % has more strong negative correlation with respect to duration of diabetes, which is more significant, ie.., FEV1 decreases with increasing duration of diabetes

FEV1/FVC & Duration of Diabetes

FEV1/FVC-R has a weak negative correlation with respect to duration of diabetes ie..,FEV1/ FVC decreases with increasing duration of diabetes. FEV1/ FVC% also has a weak negative correlation with respect to duration of diabetes.

PEF & Duration of Diabetes

PEF-R has a moderate negative correlation with respect to duration of diabetes ie.., PEF decreases with increasing duration of

diabetes. PEF% has more strong negative correlation with respect to duration of diabetes, which is more significant.

FEF 25-75% & Duration of Diabetes

FEF 25-75%-R has a moderate negative correlation with respect to duration of diabetes ie.., FEF 25-75% decreases with increasing duration of diabetes.

($FEF_{25-75\%}$) % has more strong negative correlation with respect to duration of diabetes, which is more significant.

Table 10. CORRELATION OF FBS WITH PFT VARIABLES

FVC	FVC-R	-0.49**
	FVC%	-0.691**
FEV1	FEV1-R	-0.271
	FEV1%	-0.477**
FEV1/FVC	FEV1/FVC-R	-0.088
	FEV1/FVC%	-0.087
PEF	PEF-R	-0.287
	PEF%	-0.58**
FEF 25-75%	FEF 25-75%-R	-0.525**
	FEF 25-75%(%)	-0.644**

**. Correlation is significant at the 0.01 level (2-tailed).

Effect of FBS on FVC

Both FVC & FVC% have a strong negative correlation with fasting blood sugar values, ie.., FVC & FVC% decreases as the FBS increases.

Effect of FBS on FEV1

FEV1 has a weak negative correlation than the FEV1% which has a strong negative correlation, ie., FEV1 & FEV1% decreases as FBS value increases.

Effect of FBS on FEV1/FVC

Both FEV1/FVC & FEV1/FVC% have no significant relationship with respect to the FBS values.

Effect of FBS on PEF

PEF has a weak negative correlation than the PEF% which has a strong negative correlation, ie., PEF% decreases as FBS value increases.

Effect of FBS on FEF25-75%

Both $FEF_{25-75\%}$ &($FEF_{25-75\%}$) % have a strong negative correlation with fasting blood sugar values, ie.., FVC & FVC% decreases as the FBS increases.

Table 11. CORRELATION OF PPBS WITH PFT VARIABLES

FVC	FVC-R	-0.548**
	FVC%	-0.742**
FEV1	FEV1-R	-0.319*
	FEV1%	-0.527**
FEV1/FVC	FEV1/FVC-R	-0.093
	FEV1/FVC%	-0.088
PEF	PEF-R	-0.307*
	PEF%	-0.577**
FEF 25-75%	FEF 25-75%-R	-0.606**
	FEF 25-75%(%)	-0.713**

**. Correlation is significant at the 0.01 level (2-tailed).

Effect of PPBS on FVC

Both FVC & FVC% have a strong negative correlation with fasting blood sugar values, ie.., FVC & FVC% decreases as the PPBS increases.

Effect of PPBS on FEV1

FEV1 has a moderate negative correlation than the FEV1% which has a strong negative correlation, ie., FEV1 & FEV1% decreases as PPBS value increases.

Effect of PPBS on FEV1/FVC

Both FEV1/FVC & FEV1/FVC% have no significant relationship with respect to the PPBS values.

Effect of PPBS on PEF

PEF has a moderate negative correlation than the PEF% which has a strong negative correlation, ie., PEF% decreases as PPBS value increases

Effect of PPBS on FEF25-75%

Both $FEF_{25-75\%}$ &($FEF_{25-75\%}$) % have a strong negative correlation with post prandial blood sugar values, ie.., FVC & FVC% decreases as the PPBS increases.

Table 12.CORRELATION OF HbA1c WITH PFT VARIABLES

FVC	FVC-R	-0.301*
	FVC%	-0.45**
FEV1	FEV1-R	-0.288
	FEV1%	-0.483**
FEV1/FVC	FEV1/FVC-R	-0.211
	FEV1/FVC%	-0.191
PEF	PEF-R	-0.329*
	PEF%	-0.515**
FEF 25-75%	FEF 25-75%-R	-0.339*
	FEF 25-75%(%)	-0.453**

**. Correlation is significant at the 0.01 level (2-tailed).

Effect of HbA1c on FVC

FVC has a moderate negative correlation & FVC% have a strong negative correlation with **HbA1c** levels, ie.., FVC & FVC% decreases as the HbA1c increases.

Effect of HbA1c on FEV1

FEV1 has a weak negative correlation than the FEV1% which has a strong negative correlation, ie., FEV1 & FEV1% decreases as HbA1c value increases.

Effect of HbA1c on FEV1/FVC

FEV1/FVC has a weak negative correlation & FEV1/FVC% has no significant relationship with respect to the HbA1c values.

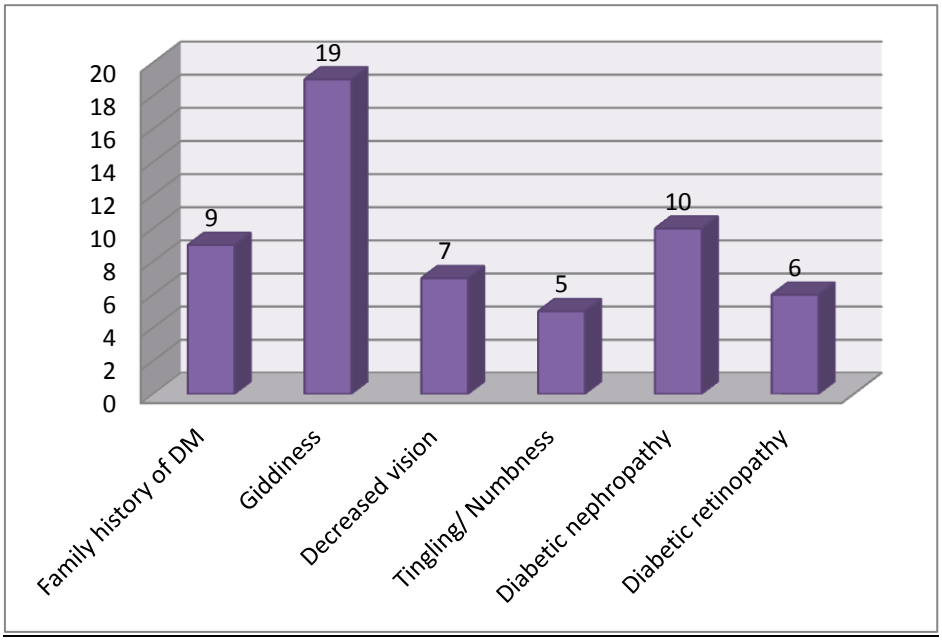
Effect of HbA1c on PEF

PEF has a moderate negative correlation than the PEF% which has a strong negative correlation, ie., PEF% decreases as HbA1c value increases

Effect of HbA1c on FEF25-75%

FEF $_{25\text{-}75\%}$ has a moderate negative correlation &(FEF $_{25\text{-}75\%}$) % have a strong negative correlation with HbA1c values, ie.., FVC & FVC% decreases as the HbA1c increases.

Graph 4. showing association of restrictive lung function with other diabetic complications



- Out of the 20 diabetic patients who had restrictive pattern lung function, 10 had diabetic nephropathy & 6 had retinopathy changes.
- 9 had family history of diabetes mellitus.
- 19 had history of giddiness, 7 with decreased vision & 5 with history of tingling sensation in both lower limbs suggestive of peripheral neuropathy.

DISCUSSION

This study was done to find out the impact of diabetes or the pulmonary functions. Two Groups namely the study & control groups were assigned with equal sex distribution along with fulfilment of inclusion and exclusion criteria already mentioned.

All the Spirometric values namely FVC, FEV1, FEV1/FVC, PEF, FEF 25-75% were having a mean decrease in diabetic group compared to the control group which was statistically proven by getting a P value of <0.05.

The % prediction of the Spirometric parameters also showed a decrease in diabetic group compared to the controls.

Among the 45 patients in diabetic groups, 14 had mild restrictive and 6 of their had moderate Spirometric pattern when analysed individually.

EFFECT OF DURATION OF DM-2 ON PULMONARY FUNCTIONS:

In the study group, it was found that the FVC%, FEV1%, PEF and PEF%, FEF 25-75% was having a strong negative correlation with the

duration of DM-2. ie., they tend to decrease with respect to increase in duration of DM-2. FEV1/FVC had only a weak negative correlation with the duration of DM-2

This Outcome was in favourable with the concept of Davis Timothy et al's study. They too showed a negative correlation of Spirometric values with the duration of Diabetes.

EFFECT OF FBS ON PULMONARY FUNCTION:

This study showed a strong negative correlation of FVC & %, FEV1%, PEF%, FEF 25-75% & its % with respect to FBS values, ie., these parameters tend to decrease with respect to increase in FBS values. But, the FEV1/FVC did not show any significant correlation.

EFFECT OF PPBS ON PULMONAY FUNCTION:

This study showed a strong negative correlation of FVC & %, FEV1%, PEF%, FEF 25-75% & % with respect to the PPBS values, ie., they tend to decrease with respect to increase in PPBS values. But, the FEV1/FVC did not show any significant correlation with respect to FBS values.

This was similar to the results of “P.Lang et al” study who showed negative correlation of spirometric values with respect of Spirometric values with respect to the glycemic status.

EFFECT OF HbA1c ON PULMONARY FUNCTION:

This study showed a strong negative correlation of FVC%, FEV1%, PEF% and FEF 25-75% with respect to the HbA1c level. ie., They tend to decrease with respect to increase in HbA1c levels which means that the Spirometric parameters decrease more in the poorly controlled diabetes patients.

5 out of 45 in the diabetic group were having mild NPDR, and they showed also mild restriction pattern in spirometry. 1 had PDR in fundus and moderate restriction in spirometry.

There results give us a clear idea that there is a prevalence of restrictive pattern of lung function in the diabetic population. Also that the Spirometric parameters are negatively correlated with respect to increase in FBS, PPBS, HbA1c values and also with respect to the duration of Diabetes Mellitus.

Out of the 20 diabetic patients who had restrictive pattern lung function, 10 had diabetic nephropathy & 6 had retinopathy changes. Which suggested the presence of microvascular complications.

The prevalence of restriction pattern of lung functions in diabetic population which is indicative of microvascular complication, makes us more cautious regarding tight control of glycemic status. Since this restriction pattern will add to the morbidity and thereby increasing the early mortality rate in diabetic patients.

CONCLUSION

1. The spirometric parameters in general were consistently lower in the diabetic group compared to the non diabetic group.
2. All the 5 parameters ie.., FVC, FEV1, FEV1/FVC, PEF, $FEF_{25-75\%}$ which were planned for the comparison showed statistically significant difference in form of decrement in diabetic group compared to the non-diabetics.
3. The pattern of spirometric parameters in diabetic group suggested the prevelance of restriction pattern in lung functions, which can be attributed to the non-enzymatic glycosylation of connective tissue resulting in the same
4. Patients with diabetic retinopathy invariably had restriction pattern in spirometry.
5. All these points to a conclusion that DIABETES MELLITUS causes decrement in lung functions in the form of restriction which is more seen in patients with longer duration of DM & patients with uncontrolled glycemic status.
6. This study adds to the literature insisting the prevalence of restriction pattern of lung function in diabetics, and hence suggesting more extensive works on this field. Probably adding diffusion studies to this can result in more clear idea of the concepts.

LIMITATIONS AND RECOMMENDATIONS

1. The study population was too small, it needs a bigger population and extensive datas to define the significant difference in a more accurate way.

2. Smoking and hypertension were the exclusion criterias., so it was difficult to collect male diabetics who were non smokers and without hypertension. Smoking was ruled out based on patients history, and there is a question of reliability.

3. Cardiac disease was ruled out on the basis of history and electrocardiogram. Echocardiogram was not done for the diabetics, so silent ischaemia in diabetics was not ruled out confidently.

4. There may be other confounding factors too which could have affected the result of this tudy, since patients were selected only based on a simple history taking and clinical examination.

5. This study did not included diffusion capacity assessment, which can give more explanation towards the restriction pattern in spirometry in the diabetics.

BIBLIOGRAPHY

1. Kasper, Braunwald, Fauci, 2004, Harrisons principles of internal medicine 17th edition vol 2, page 2286-2290

2. Marvisi M, Lino Bartolini L, del Borrello P, Brianti M, Marrani G, Guariglia A, et al. Pulmonary Function in non-insulin-dependent diabetes mellitus. Respiration 2001; 68;268-72.

3. Ljubic s, Metelko z, car N, Roglic G, Drazic Z. Reduction of diffusion capacity for carbon monoxide in diabetic patients. Chest 1998; 114:1033-5

4. King H, Albert R, Herman W. Global burden of diabetes 19952025: prevalence numerical estimates and projections. Diabetes care 1998; 21:1414-31.

5. Benbassat Carlos A, Ervin Stern, Mordechai Kramer, Joseph Lebzelter, Ilana Blum, Gershon Fink. Pulmonary function in patients with Diabetes Mellitus. The Am J Med Sci 2001; 322(3):127-132.

6. Sandler Malcom. Is the Lung a Target Organ in Diabetes Mellitus. Arch Intern Med.1990;150: 1385-1388.

7. Alvin C. Powers ;chapter 344; Harrisons Principles of Internal Medicine 18th edition.

8. Gupta R, Joshi P, Mohan V, et al. Epidemiology and causation of coronary heart disease and stroke in india. Heart 2008; 94: 16-26.

9. International Diabetes Federation Website. President of International Diabetes Federation calls for concerted action to stop Diabetes epidemic. http://www.idf.org/president-interna.

10. Ramachandran A, Mary S, Yamuna A, et al. High prevalence of diabetes of diabetes and cardiovascular risk factors associated with urbanization in India Diabetes care. 2008; 31:893-898.

11. http://www.idf.org/diabetes atlas/ 5th edition/mortality.

12. International Diabetes Federation. The IDF Diabetes Atlas. 4th edition. Brussels : International Diabetes Federation; 2009

13. Roglic G, Unwin N. Mortality attributable to diabetes: estimate for the year 2010. Diabetes Res clin Pract 2010; 87(1) : 15-19.

14. B.M.Frier, M.Fisher; Davidsons's Principles & Practice of Medicine.Chapter 21:Diabetes Mellitus.

15. Robert B Tattersall. History of diabetes mellitus, Picket and Williams Text book of Diabetes mellitus: Blackwell Scientific; 3rd edition(1) : 1-20; 2003.

16. Vasudevan D.M. , Sreekumari .S. Textbook of biochemistry. 9th edition india: Jaypee publications.

17. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New Engl J Med* 1993;329: 683-9.

18. DCCT Research Group. Effect of intensive diabetes management on macrovascular events and risk factors in the Diabetes Control and Complications Trial. *Am J Cardiol* 1995;5:894-903.

19. The Diabetes Control And Complications Trial.The Epidemiology of Diabetes Interventions and Complications (EDIC) study. *JAMA* 2003;290:2159-67.

20. DCCT Research Group. Implementation of treatment protocols in the Diabetes Control and Complications Trial. *Diabetes Care* 1995;18:361-76.

21. UK Prospective Diabetes Study Group: Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet 352*:*837*–853, *1998*

22. UK Prospective Diabetes Study Group: Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *Lancet 352*:*854*–865, *1998*

23. UK Prospective Diabetes Study Group: Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes (UKPDS 38). *BMJ 317:703*–713, *1998*

24. Ohkubo Y, Kishikawa H, Araki E, Miyata T, Isami S, Motoyoshi S, Kojima Y, *Diabetes Res Clin Pract* 28:103–117, 1995 Furuyoshi N, Shichiri M:

25. Sadikot S M, Nigam A, Das S. *et al* The burden of diabetes and impaired glucose tolerance in India using the WHO 1999 criteria: prevalence of diabetes in India study (PODIS). Diabetes Res Clin Pract 2004. 66301–307.307.

26. Pickup J, Wilham G. Epidemiology of diabetes. In: Guikshank K, ed. Textbook of diabetes. New York: Blackwell Science, 1991.

27. Bajaj M, Banerji M A. Type 2 diabetes in South Asians: a pathophysiologic focus on the Asian Indian epidemic. Curr Diab Rep 2004. 4213–218.218.

28. Ashok S, Ramu M, Deepa R. *et al* Prevalence of neuropathy in type 2 diabetes patients attending diabetes center in South India. J Assoc Physicians India 2002. 50546–550.550.

29. Levitt N S, Stansberry K B, Wychanck S. *et al* Natural progression of autonomic neuropathy and autonomic function tests in a cohort of IDDM. Diabetes Care 1996. 19751–754.754.

30. Rathmann W, Ziegler D, Jahnke M. *et al* Mortality in diabetic patients with cardiovascular autonomic neuropathy. Diabet Med 1993. 10820–824.824.

31. Pirart J. Diabetes mellitus and its degenerative complications: a prospective study of 4400 patients observed between 1947 and 1973 (third and last part). Diabetes Metab 1977. 3245–256.256.

32. Dyck P J B, Sinnreich M. Diabetic Neuropathies. Continuum 2003. 919–34.34.

33. Shy M E, Frohman E M, So Y T. *et al* Quantitative sensory testing. Neurology 2003. 602898–906.906.

34. Kennedy W R, Wendelschafer-Crabb G, Johnson T. Quantitation of epidermal nerves in diabetic neuropathy. Neurology 1996. 471042–1048.1048.

35. Consensus statement Report and recommendations of the San Antonio conference on diabetic neuropathy. American Diabetic association, American Academy of Neurology. Diabetes Care 1988. 11592–597.597.

36. Arezzo J C. The use of electrophysiology for the assessment of diabetic neuropathy. Neurosci Res Comm 1997. 2113–22.22.

37. American Diabetes Association. Standards of medical care in diabetes--2012. *Diabetes Care*. 2012 Jan;35 Suppl 1:S11-63.

38. Parving H, Mauer M, Fioretto P, Rossing P, Ritz E. Diabetic Nephropathy. *Brenner and Rector's The Kidney*. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2011: chap 38.

39. Timothy J. Barriero, D.O., and Irene Perillo, M.D., University of Rochester School of Medicine and Dentistry, Rochester, New York) *Am Fam Physician.* 2004 Mar 1;69(5):1107-1115

40. Colp CR. Interpretation of pulmonary function tests. *Chest*. 1979;76:377–8.

41. Crapo RO. Pulmonary-function testing. *N Engl J Med*. 1994; 331:25–30.

42. Petty T L. Simple office spirometry. *Clin Chest Med*. 2001; 22:845–59.

43. Margolis ML, Montoya FJ, Palma WR Jr. Pulmonary function tests: comparison of 95th percentile-based and conventional criteria of normality. *South Med J*. 1997;90:1187–91.

44. Crapo RO, Morris AH. Pulmonary function testing: sources of error in measurement and interpretation. *South Med J*. 1989;82:875–9.

45. Lung function testing: selection of reference values and interpretative strategies. American Thoracic Society. *Am J Respir Crit Care Med*. 1991; 144:1202–18.

46. P.Lange, S.Groth, J.Kastrup, M.Appleyard, J.Jansen, P.Schnohr Diabetes mellitus, plasma glucose & lung function in a cross sectional population study, Eur Resp J 1989,2.14-19.

47. Sandler M et al. Pulmonary function in IDDM with limited joint mobility. Chest 90;670,1986.

48. Uchida K et al. The findings of ventilation & perfusion scintigrams in patients with DM Resiper res.7:345,1988.

49. Schenthaner G et al. Lung elasticity in juvenile onset DM. Am Rev Respire Dis, 1977,113,37-41.

50. Jensen G et al. Epidemiology of chest pain & angina pectoris (thesis) Acta Med Scand ; 1984,suppl.682.

51. Walter.E.Robert, Alexa Beiser, Rachel J, Givelber, George T, O'Connor et al. Association between glycemic state & lung function. Am J Respir Crit Care Med 2003; 167:911-916.

52. Davis A Wendy, Mathhew knuiman, Peter kendell, Valerie Grange. Timothy M.E.Davis; Glycemic exposure is associated witj reduced pulmonary function in type 2 DM. Diabetes care 2004: 27:752-757.

53. Sandler Malcom. Is the Lung a 'Target organ' in Diabetes mellitus. Arch Intern Med.1990; 150: 1385-1388.

54. Davis Timothy M.E, Mathhew knuiman, Peter Kendell, Hein vu, Wendy A.Davis. " Reduced pulmonary function & its association

in type 2 DM: Diabetes research and clinical practice 2000; 50: 152-159.

55. Sreeja C.K, Elizabeth damuel, C.Kesava chandran, Shankar shashidharan. Pulmonary function in patients with Diabetes . IJPP 2003; 47(1): 87-93.

56. Dr.Mohan kumar, Dr.S.Arulmozhi. Diabetes in elderly: pulmonary complications in elderly diabetics. Microlabs Ltd.Bangalore 2005; 119-125.

57. Dharwadkar AR., Dharwadkar AA, Gouher banu and Shrilaxmi Bagali. Reduction in lung functions in Type 2 DM in Indian population.correlation with glycemic status. IJJP 2011; 55(2): 170-75.

58. Agarwal A.S, Fuladi A,B, Mishra G and Tayade B.O. Spirometry and diffusion studies in patients with Type 2 DM and their association with microvascular complications. 2010 vol.52. the Indian journal at chest diseases & allied sciences.

59. Hsin-chieh Yeh, phd and et al. Cross Sectional and prospective study of lung function in adults with Type 2 DM. Diabetes care 2008; 31 (4): 741-746.

60. http://faculty.quinnipiac.edu/libarts/polsci/statistics.html

LIST OF ABBREVIATIONS

IDDM	Insulin dependent diabetes mellitus
NIDDM	Non insulin dependent diabetes mellitus
FBS	Fasting blood sugar
PPBS	Post prandial blood sugar
AGE	Advanced glycated end products
HbA1c	Glycated haemoglobin
ACTH	Adreno cortico tropic hormone
NPDR	Non proliferative diabetic retinopathy
PDR	Proliferative diabetic retinopathy
ESRD	End stage renal disease
CIDP	Chronic inflammatory demyelinating polyneuropathy
TLC	Total lung capacity
FVC	Forced vital capacity
FEV1	Forced expiratory volume in 1^{st} second
$FEF_{25-75\%}$	Forced expiratory flow during middle one half of FVC
PEF	Peak expiratory flow rate
ADA	American diabetes association
JNC-7	Joint national committee-7(for Hypertension)
BMI	Body mass index
DKA	Diabetic keto-acidosis
DCCT	Diabetes Control & Complication Trial
UKPDS	United Kingdom Prospective Diabetes study

PROFORMA

NAME:

AGE/SEX:

OCCUPATION:

ADDRESS

DURATION OF DIABETES:

FAMILY H/O COPD:

TREATMENT HISTORY: 1.OHA

2.INSULIN

3.OHA+INSULIN

DIABETIC RELATED HISTORY

DECREASED VISION	
TINGLING/NUMBNESS	
BLURRING OF VISION	
GIDDINESS/BLACKOUTS	
INCREASED SWEATING	
INCONTINENCE OF URINE/FAECES	
TRANSIENT LOC	
FAMILY H/O DIABETES	
PHYSICAL EXERCISE	

GENERAL EXAMINATION:

P/Ict/Cy/Cl/LN/PE

HEIGHT: WEIGHT:

BMI:

WAIST CIRCUMFERENCE:

PULSE:

BLOOD PRESSURE----- SUPINE:

STANDING:

Cardiovascular system

Abdominal system

Respiratory system Central nervous system

INVESTIGATIONS

FBS: **FUNDOSCOPY:**

PPBS:

HbA1C:

B.UREA:

S.CREATININE:

U.MICROALBUMIN:

ECG:

CHEST X-RAY:

PULMONARY FUNCTION TEST

PARAMETERS	MEASURED	PREDICTED
FVC		
FEV1		
FEV1/FVC		
PEFR		
FEF 25%-75%		

Master chart 1. showing basic characteristics in Control

Sl no	Name	Age	Sex	Ht (mts)	Wt (kgs)	BMI	waist circumference	FBS mg/dl	PPBS mg/dl
1	bagavaan	36	M	1.72	63	21.29	88	78	136
2	ramesan	37	M	1.68	78	27.63	90	88	122
3	gopalakrishnan	38	M	1.71	72	24.62	85	76	129
4	moorthy	39	M	1.7	69	23.87	84	90	134
5	ganesh	40	M	1.72	72	24.33	76	96	138
6	dananjeyan	38	M	1.74	76	25.1	86	79	118
7	murugan	41	M	1.65	67	24.6	86	88	130
8	balasubramani	40	M	1.65	80	29.38	90	100	124
9	gopinath	42	M	1.66	70	25.4	84	98	120
10	palanivel	39	M	1.57	72	29.21	90	101	124
11	natesan	45	M	1.73	72	24.05	85	105	126
12	jayaram	46	M	1.73	63	21.04	80	95	132
13	prasath	49	M	1.75	80	26.12	83	97	130
14	ayyasamy	52	M	1.67	58	20.79	78	98	120
15	ramanujam	51	M	1.55	57	23.72	85	74	122
16	sakthi	53	F	1.64	58	21.56	86	80	126
17	Pandi	52	F	1.63	58	21.82	81	88	133
18	Sarasu	53	F	1.7	80	27.68	85	85	122
19	Ramayee	50	F	1.71	68	23.25	83	96	120
20	Rani.S	53	F	1.62	60	22.86	84	100	130
21	Rosy	51	F	1.65	79	29.01	87	90	116
22	Frabella	52	F	1.66	77	27.94	80	95	112
23	Sumathra	53	F	1.67	74	26.53	81	88	114
24	Prabha	52	F	1.66	68	24.67	82	96	130
25	Sujitha	53	F	1.6	50	19.53	83	94	98
26	Madhavi	52	F	1.65	80	29.38	80	101	133
27	Mythili.M	50	F	1.66	70	25.4	85	93	128
28	Kamala	47	F	1.54	55	23.19	82	88	124
29	Reeshma	48	F	1.52	56	24.23	84	86	126
30	Vidhya	50	F	1.51	54	23.68	84	94	122
31	Suganya	51	F	1.59	67	27.93	82	103	129
32	Abhinaya sundari	52	F	1.57	59	23.93	78	79	120
33	Chellama	53	F	1.51	68	29.82	84	80	132
34	Ruby	53	F	1.58	50	20.02	81	82	135
35	tamilarasi	53	F	1.74	85	28.07	83	88	136
36	geetha	39	F	1.58	48	19.22	81	85	130
37	vasanthi	42	F	1.47	57	21.75	85	96	128
38	kokila	41	F	1.53	53	22.64	82	93	126
39	porkodi	43	F	1.51	67	29.38	85	96	132
40	mythili	43	F	1.67	70	25.09	84	98	131
41	rani	46	F	1.58	57	22.83	86	102	132
42	ushalakshmi	50	F	1.53	56	23.92	86	78	112
43	shanthi	52	F	1.59	67	26.5	84	90	113
44	parimala	53	F	1.52	65	28.13	83	94	112
45	radha	53	F	1.73	80	26.72	85	96	111

Master chart 2. showing characterestics in Diabetic group														
Sl no	Name	Age	Sex	Duration of DM	Ht (mts)	Wt (kgs)	BMI	Waist circum (cm)	FBS mg/dl	PPBS mg/dl	Treatment History	Hb A1c	Urine albumin	Fundoscopy
1	Rajaram	47	M	9	1.59	58	22.94	84	168	230	OHA	7.3	micro	normal
2	willson	47	M	8	1.75	70	22.85	86	207	317	OHA	7.5	nil	normal
3	kanagaraj	52	M	11	1.57	60	24.34	97	240	380	OHA	7.2	macro	Mild NPDR
4	rangan	74	M	10	1.64	58	21.56	81	181	272	OHA	7.6	micro	Mild NPDR
5	murugesan	64	M	12	1.58	63	25.23	96	190	286	OHA	7.7	micro	Mild NPDR
6	narayanasami	67	M	20	1.68	60	21.25	86	175	280	OHA	6.8	nil	normal
7	kuppan	48	M	7	1.56	60	24.65	88	120	152	OHA	6.9	nil	normal
8	vasanthan	60	M	6	1.54	58	24.45	90	112	130	OHA	6.6	nil	normal
9	sivakumar	55	M	12	1.58	62	24.83	92	167	268	OHA	7.2	micro	normal
10	mani	48	M	6	1.61	63	24.3	88	102	145	OHA	6.5	nil	normal
11	kamalakannan	51	M	7	1.72	70	23.66	89	156	189	OHA	7.4	micro	normal
12	arumugam	49	M	7	1.74	68	22.46	94	110	130	OHA	6.9	nil	normal
13	murugaiya	48	M	7	1.68	67	23.73	91	106	146	OHA	7	nil	normal
14	magesan	48	M	6	1.56	60	24.65	86	106	123	OHA	7.1	nil	normal
15	ravi	50	M	7	1.72	58	19.6	88	123	150	OHA	6.9	nil	normal
16	Rani	55	F	13	1.52	63	27.2	83	188	260	OHA+INSULIN	7.8	macro	Mild NPDR
17	jothi	52	F	12	1.61	65	25.07	85	170	268	OHA	6.9	nil	normal
18	sharon	47	F	7	1.45	51	24.25	80	198	287	OHA	6.8	nil	normal
19	saraswathi	45	F	7	1.55	63	26.22	80	181	280	OHA	6.7	nil	normal
20	lutmary	50	F	6	1.5	65	28.88	82	85	130	OHA	6.5	nil	normal
21	jayalaxmi	51	F	9	1.58	72	28.84	91	128	180	OHA	6.6	nil	normal
22	renuka	47	F	8	1.5	63	28	95	148	200	OHA	7	nil	normal
23	rajeshwari	39	F	6	1.51	62	27.19	83	130	206	OHA	7.2	nil	normal
24	girija	51	F	6	1.44	49	23.63	81	156	190	OHA	7.1	nil	normal
25	vijayalaxmi	48	F	13	1.5	62	27.55	90	182	290	OHA	7.4	nil	normal
26	indira	46	F	6	1.47	64	29.61	94	150	196	OHA	7.2	nil	normal
27	govindammal	42	F	8	1.49	55	24.77	78	170	228	OHA	7.3	nil	normal
28	parasakthi	50	F	12	1.45	58	27.58	88	190	386	OHA+INSULIN	7.2	macro	Mild NPDR
29	kalaivani	44	F	9	1.49	61	27.47	91	114	178	OHA	7.1	nil	normal
30	padmavathy	68	F	10	1.55	60	24.9	95	106	124	OHA	6.8	nil	normal
31	parvathy	51	F	6	1.46	47	22.04	82	99	130	OHA	6.6	nil	normal
32	kottiswari	42	F	15	1.53	63	26.91	91	260	320	OHA+INSULIN	8	macro	Moderate NPDR
33	rajam	63	F	7	1.47	53	24.52	81	155	187	OHA	7.1	nil	normal
34	muniyamma	55	F	7	1.53	62	26.48	93	130	195	OHA	6.8	micro	normal
35	hamsa	39	F	6	1.63	77	28.98	95	140	166	OHA	6.9	nil	normal
36	siddiqa	63	F	7	1.53	62	26.48	92	129	200	OHA	7.3	nil	normal
37	thenmozhi	50	F	12	1.5	55	24.44	85	230	367	OHA	7.1	nil	normal
38	sembammal	58	F	9	1.45	50	23.78	86	168	260	OHA	7.2	nil	normal
39	malliga	47	F	11	1.52	43	18.61	66	156	248	OHA	7.1	nil	normal
40	savithri.s	53	F	8	1.55	60	24.97	98	161	257	OHA	7.2	nil	normal
41	kaliamma	52	F	9	1.51	55	24.12	85	192	280	OHA	7.5	micro	normal
42	nagapandi	64	F	6	1.51	55	24.12	84	102	135	OHA	6.5	nil	normal
43	sarasamma	52	F	8	1.52	57	24.67	87	127	146	OHA	7.1	nil	normal
44	logeswari	45	F	7	1.5	55	24.44	85	110	127	OHA	6.5	nil	normal
45	balamma	58	F	12	1.52	60	25.96	89	166	230	OHA	8	micro	normal

Master chart 3. showing spirometry results in control group

Sl No	Name	Spirometer ID	FVC		%	FEV1		%	FEV1/FVC		%	PEF		%	FEF 25-75%		%	Interpretation
			Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		
1	bagavaan	1703	3.47	4.03	86	3.13	3.36	93	90.2	80.7	112	7.77	9.16	85	4.56	4.49	102	Normal
2	ramesan	1706	3.36	3.81	88	3.07	3.18	97	91.4	80.6	113	7.47	8.87	84	4.45	4.37	104	Normal
3	gopalakrishnan	1711	4.17	3.93	106	3.21	3.27	98	77	80.4	96	8.02	9.02	89	4.9	4.38	112	Normal
4	moorthy	1715	3.94	3.86	102	3.32	3.21	103	84.3	80.2	105	8.23	8.91	92	2.82	4.32	65	Normal
5	ganesh	1723	3.65	3.94	93	3.36	3.26	103	92.1	80	115	8.34	8.99	93	5.36	4.32	124	Normal
6	dananjeyan	1729	3.56	4.08	87	3.31	3.38	98	93	80.4	116	7.91	9.2	86	5.23	4.44	118	Normal
7	murugan	1735	3.61	3.57	101	3.33	2.98	112	92.2	79.8	116	7.54	8.52	88	5.01	4.14	121	Normal
8	balasubramani	1737	3.68	3.58	103	3.36	3	112	91.3	80	114	7.74	8.56	90	4.97	4.18	119	Normal
9	gopinath	1738	3.67	3.59	102	3.38	2.98	113	92.1	79.7	116	6.89	8.54	81	5.24	4.11	127	Normal
10	palanivel	1742	3.41	3.21	106	3.15	2.72	116	92.4	80.2	115	7.29	8.11	90	4.87	4.07	120	Normal
11	natesan	1746	3.59	3.88	93	3.27	3.17	103	91.1	79.1	115	6.72	8.84	76	4.85	4.12	118	Normal
12	jayaram	1750	3.41	3.85	89	3.21	3.15	102	94.1	78.9	119	6.23	8.79	71	5.17	4.08	127	Normal
13	prasath	1753	3.52	3.89	90	3.19	3.14	102	90.6	78.4	116	6.9	8.79	78	4.76	3.99	119	Normal
14	ayyasamy	1760	3.47	3.31	105	3.21	2.7	119	92.5	77.9	119	6.14	8.05	76	4.82	3.67	131	Normal
15	ramanujam	1764	3.66	2.84	129	3.36	2.35	143	91.8	78	118	6.5	7.47	87	5.08	3.51	145	Normal
16	sakthi	1766	2.86	2.6	110	2.58	2.22	116	90.2	79	114	5.2	6.32	82	3.86	3.17	122	Normal
17	pandi	1769	2.94	2.59	114	2.69	2.21	122	91.5	79.2	116	4.92	6.3	78	3.97	3.19	124	Normal
18	sarasu	1785	2.4	2.84	85	2.2	2.43	91	91.7	79	116	3.96	6.65	60	3.33	3.24	103	Normal
19	ramayee	1790	2.74	2.94	93	2.52	2.52	100	92	79.6	116	5.52	6.8	81	4.03	3.35	120	Normal
20	rani.S	1805	2.63	2.53	104	2.42	2.15	113	92	79	116	4.33	6.21	70	3.55	3.14	113	Normal
21	rosy	1807	2.41	2.69	90	2.27	2.3	99	94.2	79.4	119	5.06	6.44	79	3.65	3.25	112	Normal
22	frabella	1811	2.67	2.71	99	2.39	2.31	103	89.5	79.2	113	3.96	6.46	61	3.34	3.22	104	Normal
23	sumathra	1816	2.42	2.72	89	2.19	2.32	94	90.5	79	115	3.66	6.49	56	2.95	3.2	92	Normal
24	prabha	1821	2.59	2.71	96	2.34	2.31	101	90.3	79.2	114	3.52	6.46	54	3.01	3.22	93	Normal
25	sujitha	1826	2.29	2.45	93	2.01	2.08	97	87.8	79	111	2.98	6.1	49	2.49	3.12	80	Normal
26	madhavi	1835	2.44	2.66	92	2.27	2.27	100	93	79.2	117	4.01	6.41	63	3.39	3.21	106	Normal
27	mythili.M	1837	2.34	2.75	85	2.12	2.35	90	90.6	79.6	114	4.68	6.52	72	3	3.29	91	Normal
28	kamala	1839	2.59	2.36	110	2.33	2.01	116	90	80	112	4.22	5.95	71	3.32	3.24	102	Normal
29	reeshma	1842	2.5	2.25	111	2.23	1.91	117	89.2	80	112	3.83	5.81	66	2.78	3.19	87	Normal
30	vidhya	1846	2.03	2.18	93	1.81	1.84	98	89.2	79.6	112	2.71	5.7	48	2.21	3.1	71	Normal
31	suganya	1850	2.3	2.46	93	2.06	2.09	99	89.6	79.4	113	3.07	6.11	50	2.67	3.17	84	Normal
32	abhinaya sundari	1852	2.32	2.36	98	2.08	2	104	89.7	79.2	113	3.26	5.97	55	2.64	3.11	85	Normal
33	chellama	1857	1.98	2.11	94	1.71	1.77	97	86.4	79	109	2.43	5.61	43	1.94	3	65	Normal
34	ruby	1861	2.07	2.38	87	1.88	2.01	94	90.8	79	115	2.89	5.99	48	2.35	3.09	76	Normal
35	tamilarasi	1864	2.61	2.99	87	2.39	2.57	93	91.6	79	116	5.11	6.87	74	3.62	3.29	110	Normal
36	geetha	1869	2.28	2.69	85	2.08	2031	90	91.2	81.7	112	4.64	6.41	72	3.17	3.57	89	Normal
37	vasanthi	1871	2.21	2.2	100	2.04	1.87	109	92.3	81.1	114	4.13	5.72	72	2.99	3.33	90	Normal
38	kokila	1873	2.21	2.45	90	1.93	2.11	91	87.3	81.3	107	3.06	6.08	50	2.25	3.44	65	Normal
39	porkodi	1876	2.29	2.33	98	2.1	1.99	106	91.7	80.9	113	4.14	5.91	70	2.79	3.34	84	Normal
40	mythili.M	1879	2.57	2.95	87	5.35	2.54	93	91.4	80.9	113	4.19	6.79	62	3.38	3.54	95	Normal
41	rani	1880	2.21	2.53	87	2.03	2.17	94	91.9	80.4	114	4.29	6.2	69	3.07	3.33	92	Normal
42	ushalakshmi	1886	2.07	2.25	92	1.87	1.91	98	90.3	79.6	113	3.62	5.81	62	2.52	3.13	81	Normal
43	shanthi	1889	2.15	2.44	88	1.94	2.07	94	90.2	79.2	114	3.62	6.08	60	2.76	3.14	88	Normal
44	parimala	1894	1.83	2.14	86	1.67	1.81	92	91.3	79	116	3.68	5.66	65	2.41	3.02	80	Normal
45	radha	1901	2.89	2.95	98	2.67	2.53	106	92.4	79	117	5.19	6.82	76	3.91	3.28	119	Normal

Sl No	Name	spirometer Lab ID	FVC		%	FEV1		%	FEV1/FVC		%	PEF		%	PEF 25-75%		%	Interpretation
			Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		Recorded	Predicted		
1	rajaram	1292	2.49	3.13	79	2.02	2.59	78	79.8	78.8	101	2.53	7.89	32	2.08	3.76	55	mild restriction
2	uillran	1232	2.96	3.93	75	2.49	3.19	78	84.1	78.8	107	5.95	8.87	67	2.6	4.07	64	mild restriction
3	kanagaraj	1237	1.52	2.94	52	3.33	2.42	138	85.4	78	109	6.81	7.6	31	1.03	3.55	29	moderate restriction
4	rangan	1262	2.07	2.77	75	1.67	2.11	79	80.7	73.9	109	3.1	7.04	44	1.7	2.7	63	mild restriction
5	muruganan	1263	1.84	2.7	68	1.45	2.13	68	78.8	75.7	104	3.31	7.1	47	1.58	3.01	52	moderate restriction
6	narayanasami	1285	2.62	3.38	78	2.34	2.71	86	89.3	77.1	116	4.65	8.06	58	2.16	3.55	61	mild restriction
7	kuppan	1274	2.37	2.41	98	2.14	2.05	104	90.3	80	113	4.31	6.03	71	2.96	3.24	91	normal
8	varanthan	1288	2.53	2.58	98	2.26	2.03	109	89.3	79.4	117	5.48	7.03	78	3.16	3.11	102	normal
9	sivakumar	1290	2.23	2.9	77	1.94	2.36	82	87	77.3	113	3.53	7.49	47	2.32	3.4	68	mild restriction
10	mani	1298	2.91	3.21	91	2.63	2.64	100	90.4	78.6	115	6.49	7.97	81	3.83	3.76	102	normal
11	kamalakannan	1356	2.81	3.69	76	2.59	2.98	87	92.2	78	118	5.66	8.52	66	3.9	3.84	102	normal
12	arumugam	1333	3.36	3.84	88	3.14	3.11	101	93.5	78.4	119	7.81	8.73	89	4.8	3.97	121	normal
13	murugaiya	1365	3.05	3.56	86	2.77	2.91	95	90.8	78.6	116	6.61	8.4	79	4.23	3.9	108	normal
14	nageran	1231	2.36	2.41	98	2.15	2.05	104	91	80	112	4.29	6.03	72	2.89	3.24	91	normal
15	ravi	1456	3.21	3.71	87	2.95	3.01	98	91.9	78.2	118	7.34	8.56	86	4.72	3.89	121	normal
16	rani	1233	1.41	2.1	67	1.16	1.77	66	82.3	78.7	105	1.6	5.6	29	1.19	2.95	40	moderate restriction
17	jothi	1282	1.86	2.53	74	1.61	2.16	75	86.6	79.4	109	2.92	6.22	47	1.98	3.2	62	mild restriction
18	sharan	1281	1.5	2.01	75	1.27	1.7	75	84.7	80.2	106	2.18	5.46	40	1.42	3.13	45	mild restriction
19	rararuathi	1283	1.73	2.44	71	1.55	2.08	75	89.6	80.6	111	3.31	6.07	55	1.97	3.32	59	mild restriction
20	lutmary	1245	2.38	2.13	112	2.12	1.8	118	89.7	79.6	112	4.71	5.64	84	3.05	3.09	99	normal
21	jayalaxmi	1265	2.13	2.42	88	1.98	2.05	97	93	79.4	117	4.84	6.05	80	2.96	3.16	94	normal
22	renuka	1268	2.05	2.2	93	1.87	1.87	100	91.2	80.2	114	4.8	5.73	84	2.85	3.19	89	normal
23	rajeruari	1253	2.14	2.42	88	1.87	2.08	90	87.4	81.7	107	4.19	6.03	69	1.98	3.48	57	normal
24	girija	1258	1.95	1.88	104	1.79	1.57	114	91.8	79.4	116	4.68	5.28	89	2.78	2.98	93	normal
25	vijayalaxmi	1255	1.72	2.18	79	1.48	1.84	80	86	80	108	2.02	5.7	35	1.76	3.16	56	mild restriction
26	indira	1256	1.87	2.1	89	1.73	1.78	97	92.5	80.4	115	2.64	5.6	47	1.95	3.19	61	normal
27	govindammal	1252	2.65	2.28	116	2.16	1.94	111	81.5	81.1	100	5.91	5.83	51	2.32	3.35	69	normal
28	pararakthi	1254	1.13	1.94	58	0.95	1.64	58	84.1	79.6	106	3	5.37	56	1.07	3.03	35	moderate restriction
29	kalaivani	1251	2.1	2.25	93	1.87	1.92	97	89	80.9	110	4.6	5.8	79	2.33	3.32	70	normal
30	padmavathy	1243	2.86	1.92	149	1.44	1.58	91	50.3	76.2	66	2.09	5.38	37	1.28	2.54	50	normal
31	parvathy	1239	1.64	1.96	84	1.45	1.64	88	88.4	79.4	111	2.64	5.39	49	1.82	3.01	60	normal
32	kattiruari	1241	1.67	2.43	69	1.4	2.08	67	83.8	81.1	103	2.55	6.05	42	1.64	3.4	48	moderate restriction
33	rajam	1240	1.63	1.72	95	1.61	1.42	113	98.8	77.1	128	2.38	5.09	47	2.33	2.61	89	normal
34	muniyamma	1238	2.25	2.14	105	1.97	1.8	109	87.6	78.7	111	2.37	5.66	42	1.84	2.96	62	normal
35	hamra	1231	2.56	2.88	89	2.29	2.49	92	89.5	81.7	110	4.41	6.69	66	3.28	3.63	90	normal
36	ziddiqa	1234	1.69	1.96	86	1.42	1.63	87	84	77.1	109	2.24	5.42	41	1.48	2.69	55	normal
37	thenmozhi	1294	1.2	2.13	56	1.15	1.8	64	95.8	79.6	120	2.95	5.64	52	1.33	3.09	43	moderate restriction
38	rombammal	1293	1.27	1.74	73	1.22	1.44	85	96.1	77.9	123	3.11	5.1	61	2.65	2.72	97	mild restriction
39	malliga	1291	1.8	2.31	78	1.56	1.96	80	86.7	80.4	108	3.01	5.87	51	1.91	3.25	59	mild restriction
40	ravithris	1297	1.66	2.66	73	1.43	1.91	75	86.1	79	109	2.87	5.83	49	1.98	3.05	65	mild restriction
41	kaliamma	1304	1.61	2.12	76	1.4	1.79	78	87	79.2	110	2.83	5.64	50	1.71	3.04	56	mild restriction
42	nagapandi	1311	1.94	1.85	105	1.68	1.53	110	86.6	76.9	113	3.41	5.28	65	2.02	2.63	77	normal
43	rararamma	1357	1.89	2.17	87	1.75	1.83	96	92.6	79.2	117	4.91	5.69	86	5.62	3.05	86	normal
44	lageruari	1410	1.96	2.24	88	1.8	1.91	94	91.8	80.6	114	5.13	5.79	89	2.72	3.26	83	normal
45	balamma	1433	1.47	2.03	72	1.22	1.7	72	83	78.1	106	1.84	5.51	33	1.27	2.85	45	mild restriction

Sl.no	name	Famiy h/o Diabetes	blurring of vision	giddiness/ blackout	tingling/ numbness	inreased sweating	transient LOC	Physical exercise	Family h/o COPD
1	rajaram	NO	YES	YES	NO	NO	NO	NO	NO
2	willson	NO	NO	YES	NO	NO	NO	NO	NO
3	kanagaraj	NO	YES	YES	YES	NO	NO	NO	NO
4	rangan	NO	NO	YES	NO	NO	NO	NO	NO
5	murugesan	YES	NO	YES	NO	NO	NO	NO	NO
6	narayanasami	YES	NO	YES	NO	NO	NO	NO	NO
7	kuppan	NO	NO	NO	NO	NO	NO	YES	NO
8	vasanthan	NO	NO	NO	NO	NO	NO	YES	NO
9	sivakumar	NO	YES	YES	NO	NO	NO	NO	YES
10	mani	YES	NO	NO	NO	NO	NO	YES	NO
11	kamalakannan	YES	NO	YES	NO	NO	NO	NO	NO
12	arumugam	YES	NO	YES	NO	NO	NO	YES	NO
13	murugaiya	NO	NO	NO	NO	NO	NO	YES	NO
14	magesan	YES	YES	YES	NO	NO	NO	YES	NO
15	ravi	YES	NO	YES	NO	NO	NO	YES	NO
16	rani	YES	NO	YES	YES	YES	NO	NO	YES
17	jothi	YES	NO	YES	NO	NO	NO	YES	NO
18	sharon	YES	NO	YES	NO	NO	NO	NO	NO
19	saraswathi	YES	NO	YES	NO	NO	NO	YES	NO
20	lutmary	YES	NO	NO	NO	NO	NO	YES	NO
21	jayalaxmi	YES	YES	YES	NO	NO	NO	NO	NO
22	renuka	YES	NO	NO	NO	NO	NO	NO	NO
23	rajeswari	NO	NO	NO	NO	NO	NO	NO	NO
24	girija	YES	NO	YES	NO	NO	NO	NO	NO
25	vijayalaxmi	NO	NO	YES	NO	NO	NO	NO	NO
26	indira	YES	NO	YES	NO	NO	NO	YES	NO
27	govindammal	YES	NO	YES	NO	NO	NO	YES	NO
28	parasakthi	YES	YES	YES	YES	YES	NO	NO	NO
29	kalaivani	YES	NO	YES	NO	NO	NO	YES	NO
30	padmavathy	YES	NO	YES	NO	NO	NO	NO	NO
31	parvathy	NO	NO	NO	NO	NO	NO	YES	NO
32	kottiswari	YES	YES	YES	YES	YES	NO	NO	NO
33	rajam	YES	NO	YES	NO	NO	NO	NO	NO
34	muniyamma	YES	NO	YES	NO	NO	NO	YES	NO
35	hamsa	NO	YES	YES	NO	NO	NO	NO	NO
36	siddiqa	YES	NO	YES	NO	NO	NO	YES	NO
37	thenmozhi	NO	YES	YES	NO	NO	NO	NO	NO
38	sembammal	NO	NO	NO	NO	NO	NO	NO	NO
39	malliga	YES	NO	YES	NO	NO	NO	YES	NO
40	savithri.s	NO	NO	YES	NO	NO	NO	NO	NO
41	kaliamma	YES	NO	YES	NO	NO	NO	NO	NO
42	nagapandi	YES	NO	NO	NO	NO	NO	YES	NO
43	sarasamma	NO	NO	YES	NO	NO	NO	NO	NO
44	logeswari	YES	NO	NO	NO	NO	NO	YES	NO
45	balamma	NO	YES	YES	YES	NO	NO	NO	NO

ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
CHENNAI- 10.
Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10

Sub: Ethical Committee project work - approved – regarding.

Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

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With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

S.NO.	Name	Topic
1.	Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai.	1.To Identify a Safe Zone to approach proximal Humerus 2.To study Anatomical relations of Axillary nerve, its course & its Variations
2.	Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai	Heriditary Multiple Exostosis
3.	Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10	Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT
4.	Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10	Cord Blood Zinc Level in Term-Small for Gestational Age Neonates
5.	Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai	Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis
6.	R.Prathiban,(Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32	Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus
7.	B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects
8.	G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects

9.	R. Ragulji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32.	A Study of Pulmonary function in insulin dependent diabetes mellitus
10.	V.M. Jenila Veny,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32	Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease
11.	Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10	A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding
12.	Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai	Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy
13.	Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients
14.	Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss
15.	Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients
16.	Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai	Dyslipidemia as a Predictor of PIH
17.	Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32.	Association of subclinical hypothyroidism in metabolic syndrome patients
18.	Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus
19.	Dr.B.Priya, MD(G.M.), PG	Study of Serum Bilirubin Concentration in Established Coronary Artery Disease
20.	Dr.R.Hema, MD(G.M.), PG.,	Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients
21.	Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Pulmonary Functions in Type 2 Diabetes Mellitus
22.	Dr.M.Dhanasekar, MD(G.M.), PG.,	Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien
23.	Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10	A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy

24.	Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study of Stress Hyperglycemia in Moderate Degree Burns
25	Dr. V. Nandakumar, MD(G.M.), PG.,	A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus
26.	Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai	A Study of Unusual Presentations of Appendicitis.
27.	Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai	A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer
28.	Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai.	Wound healing efficacy of the chitosan – containing collagenous biomaterial, on burn wound
29.	E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur.	Isolation and Characterization of Bacterial Pathogens from Eye Infection

We are glad to inform you that at the Ethical Committee meeting, the document were discussed and the above short term projects are Ethically approved.

4/2/11

CHAIRPERSON

DEAN

Govt. Kilpauk Medical College,

Chennai-10.

To: The Individuals